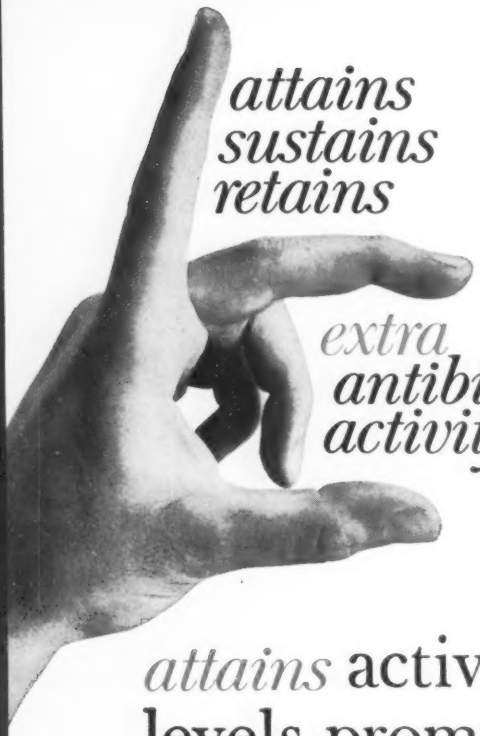
VIRGINIA MEDICAL MONTHLY

OFFICIAL PUBLICATION OF THE MEDICAL SOCIETY OF VIRGINIA

FEBRUARY, 1961



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sustains
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extra
antibiotic
activity

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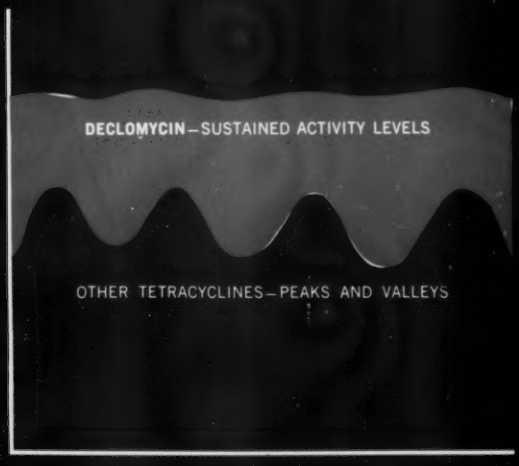

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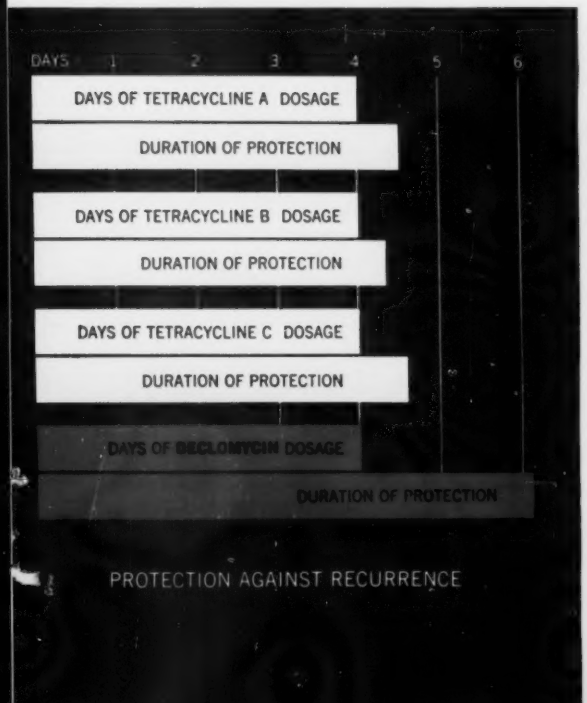
DAYS 1 2 3 4 5 6
DAYS OF TETRACYCLINE A DOSAGE
DURATION OF PROTECTION
DAYS OF TETRACYCLINE B DOSAGE
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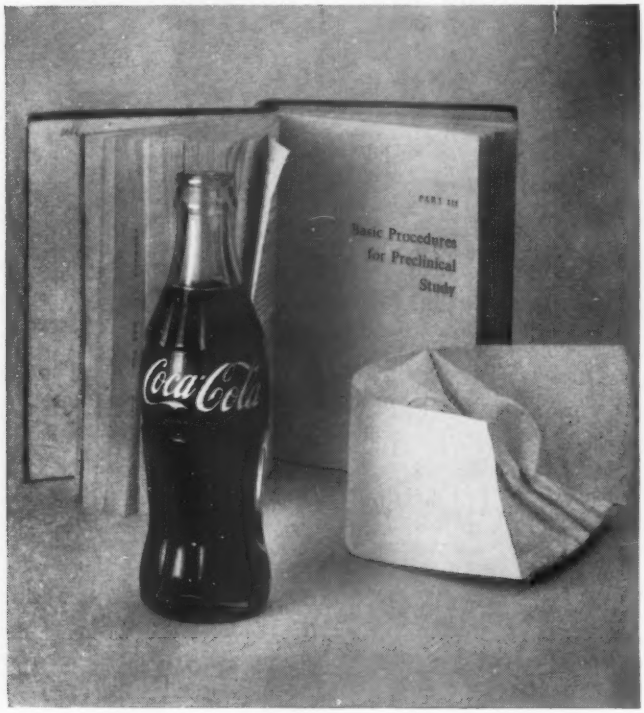

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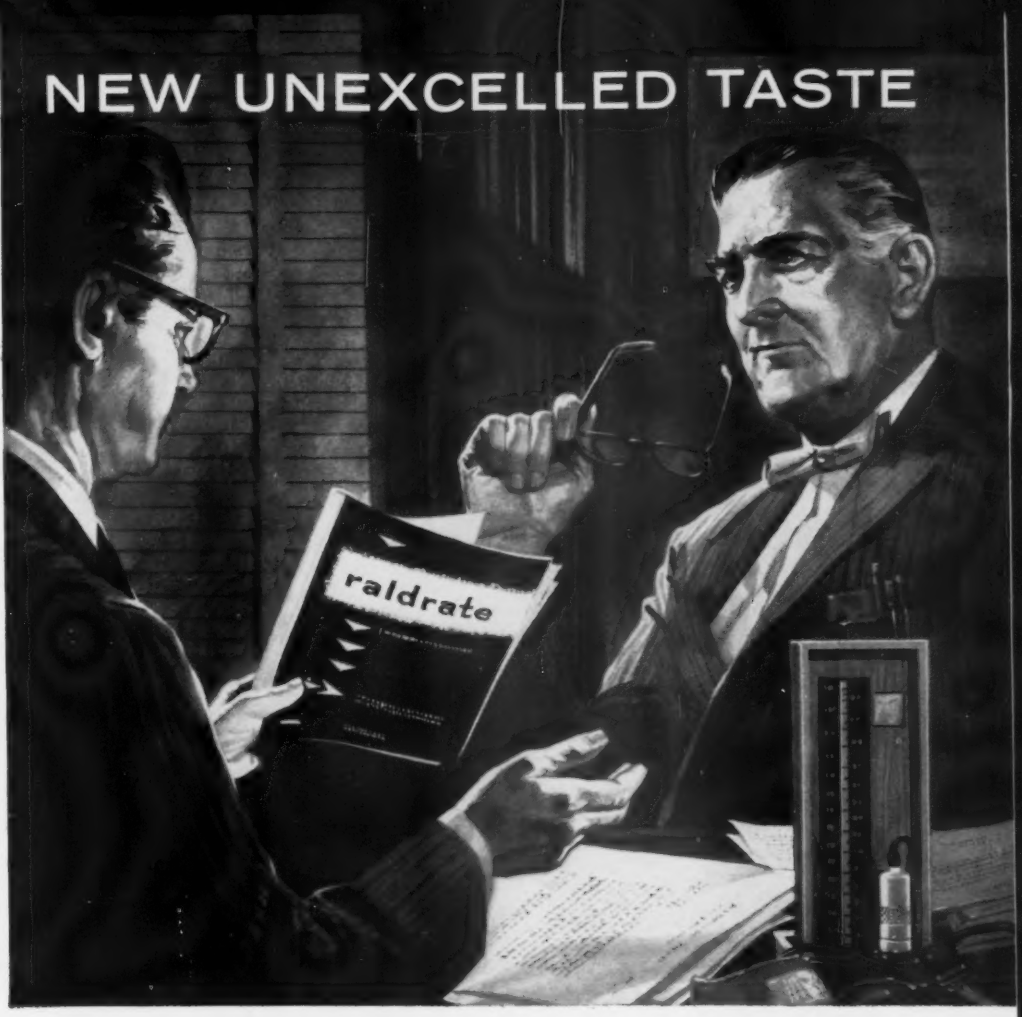

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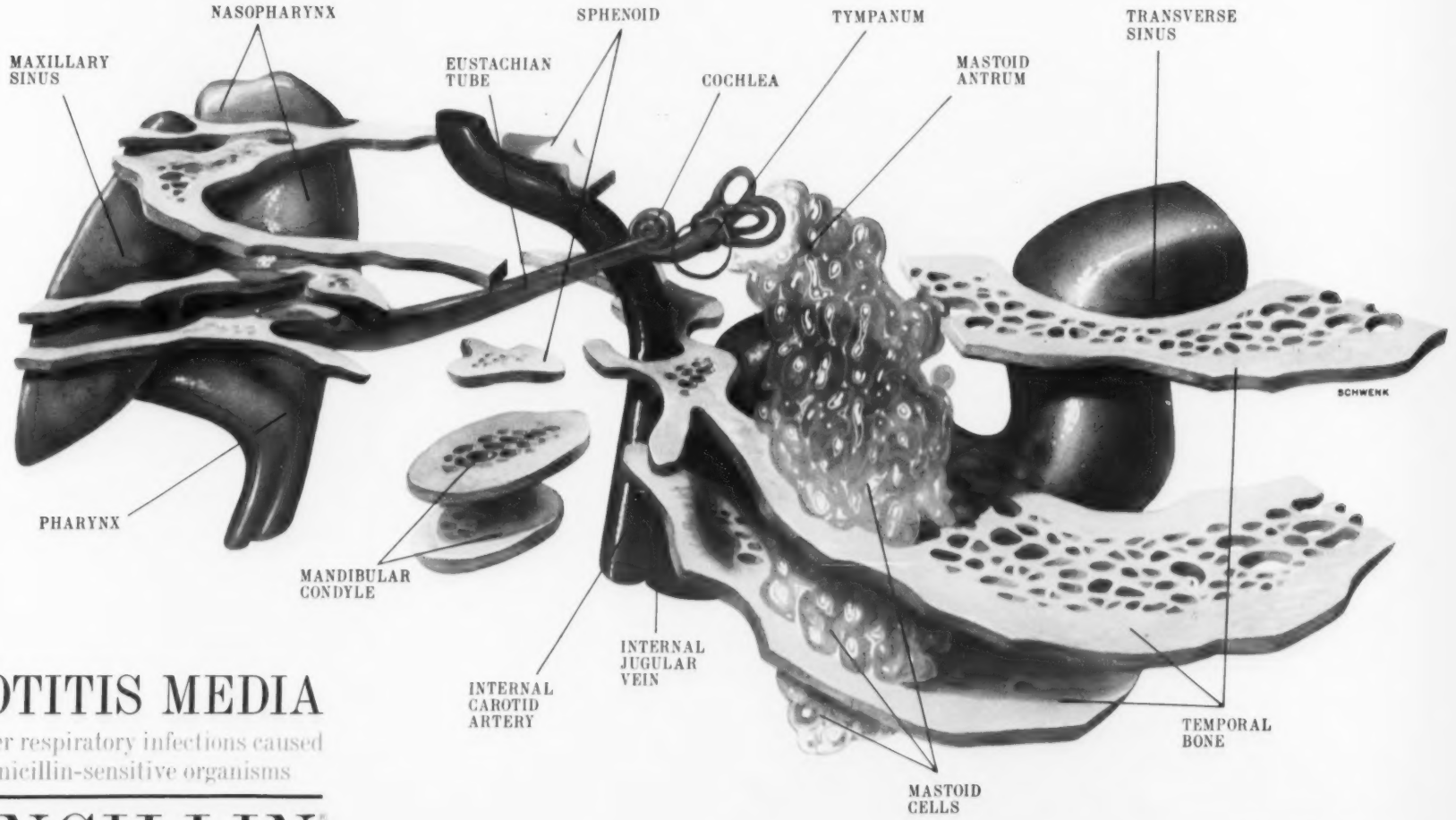

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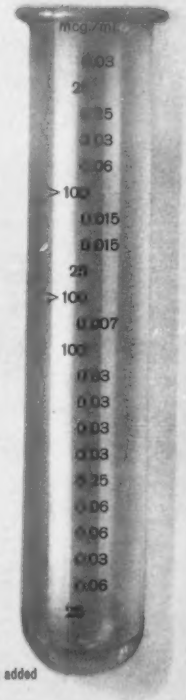

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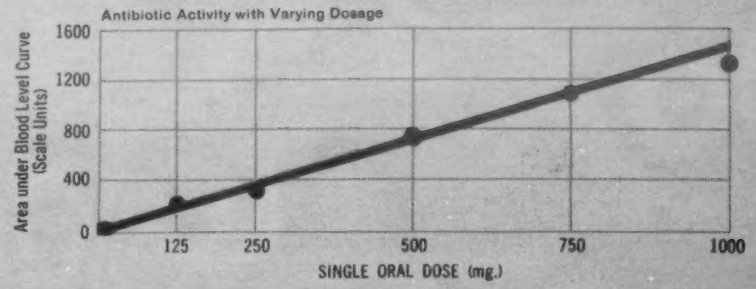

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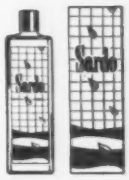
10 Ichthyosis	3	4	3
Skin Conditions	Benefited	No Benefit	
20 Nummular dermatitis	19	1	
10 Neurodermatitis	10	–	



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1. Weissberg, G.: Clin. Med., June 1960.

2. Spoor, H. J.: N. Y. St. J. Med., Oct. 15, 1958.

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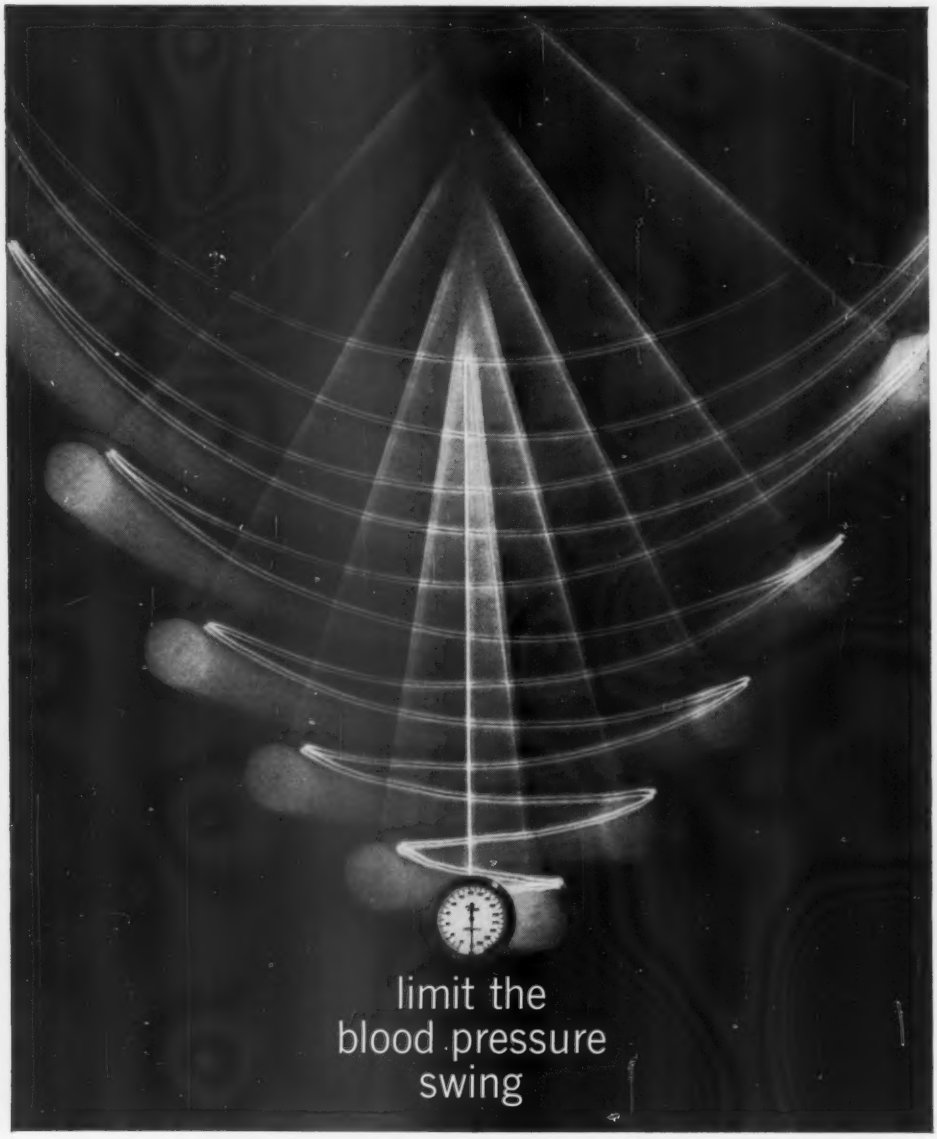

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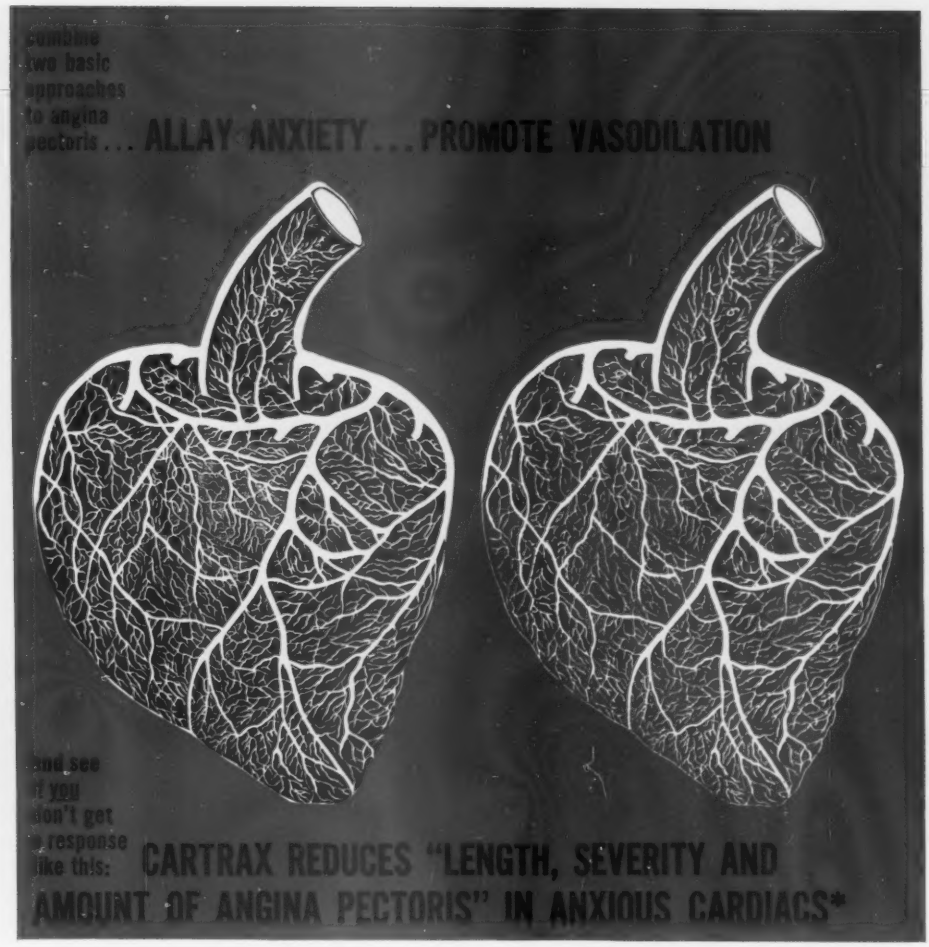

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*Council on Drugs, J.A.M.A. 165:58 (Sept. 7) 1957.

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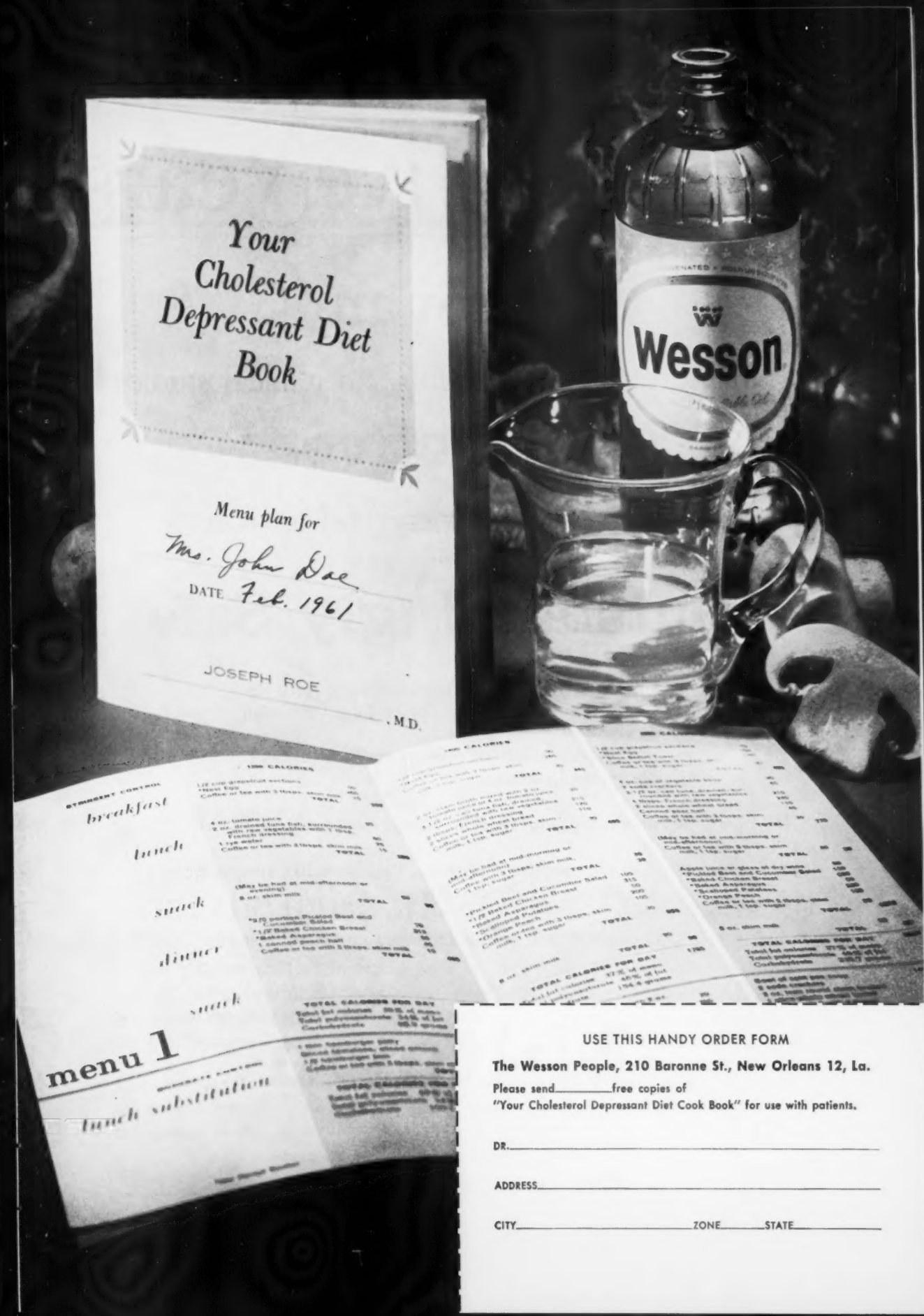

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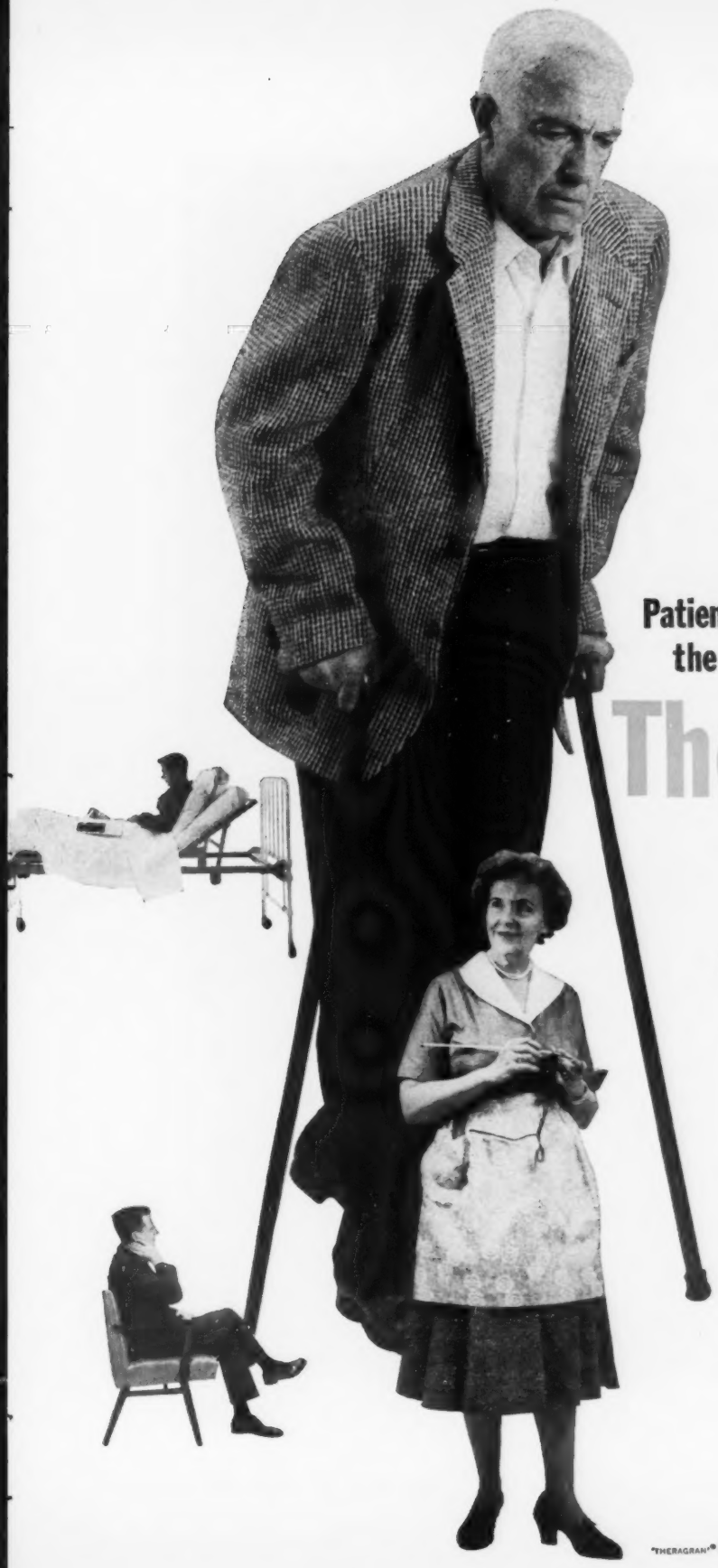

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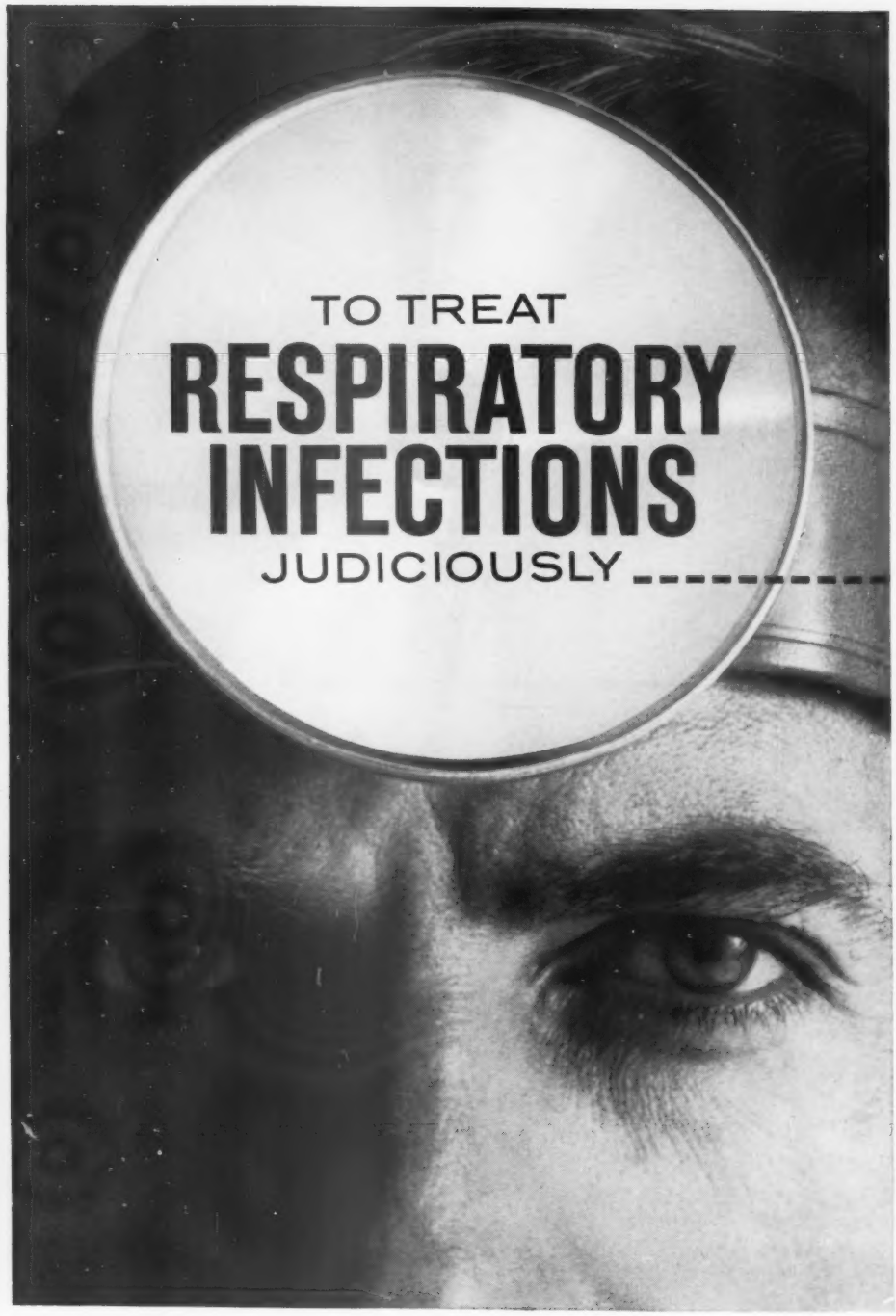
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Ephedrine sulfate	12 mg.
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Luminal (brand of phenobarbital)	6 mg.
Alcohol	19%

Dosage.
Children—from ½ to 3 teaspoons (5 to 15 cc.) three times daily as required. Adults—2 or 3 tablespoons (15 to 30 cc.) three or four times daily as required.

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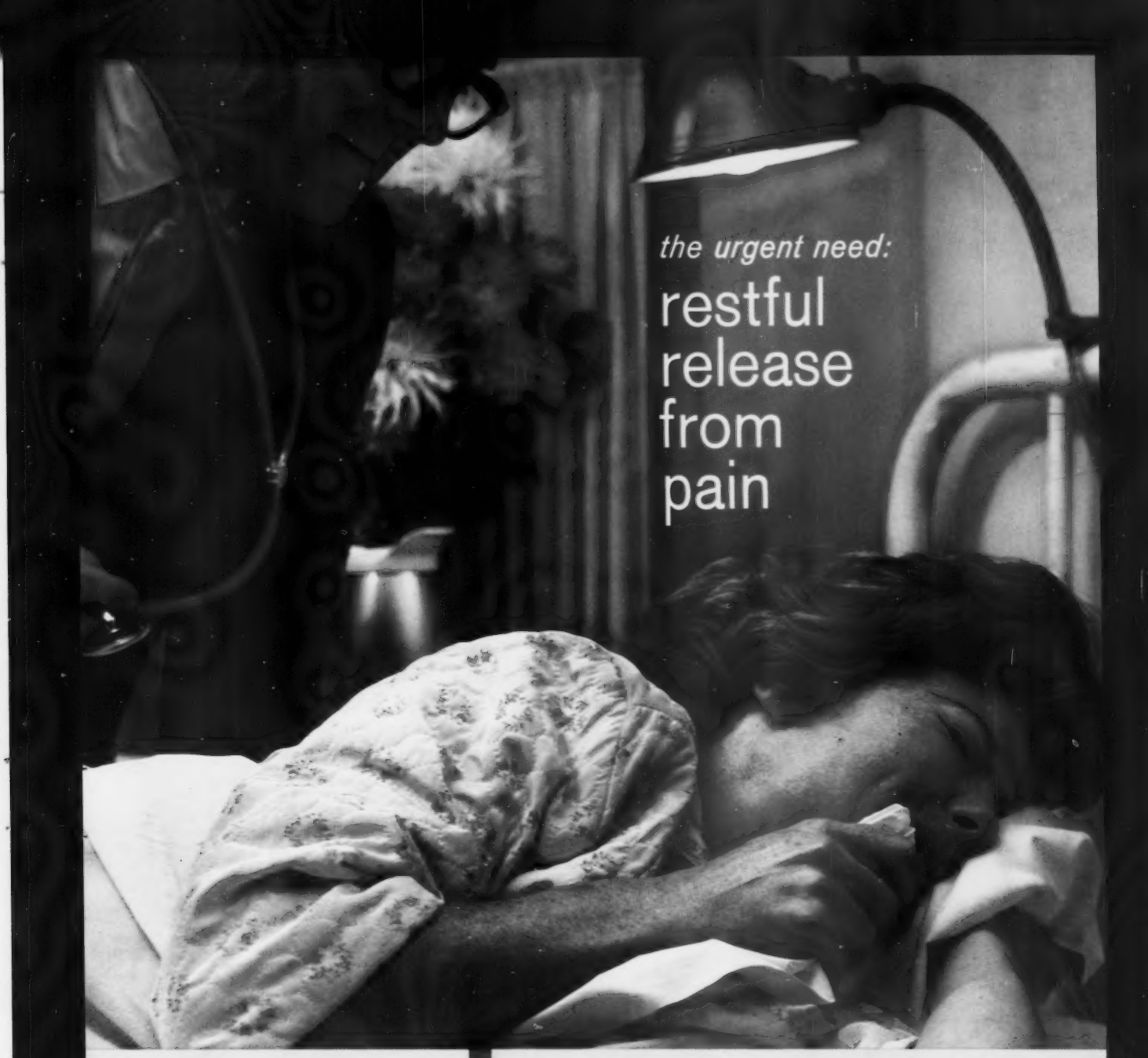

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PHENAPHEN with Codeine ..... ¼ gr.

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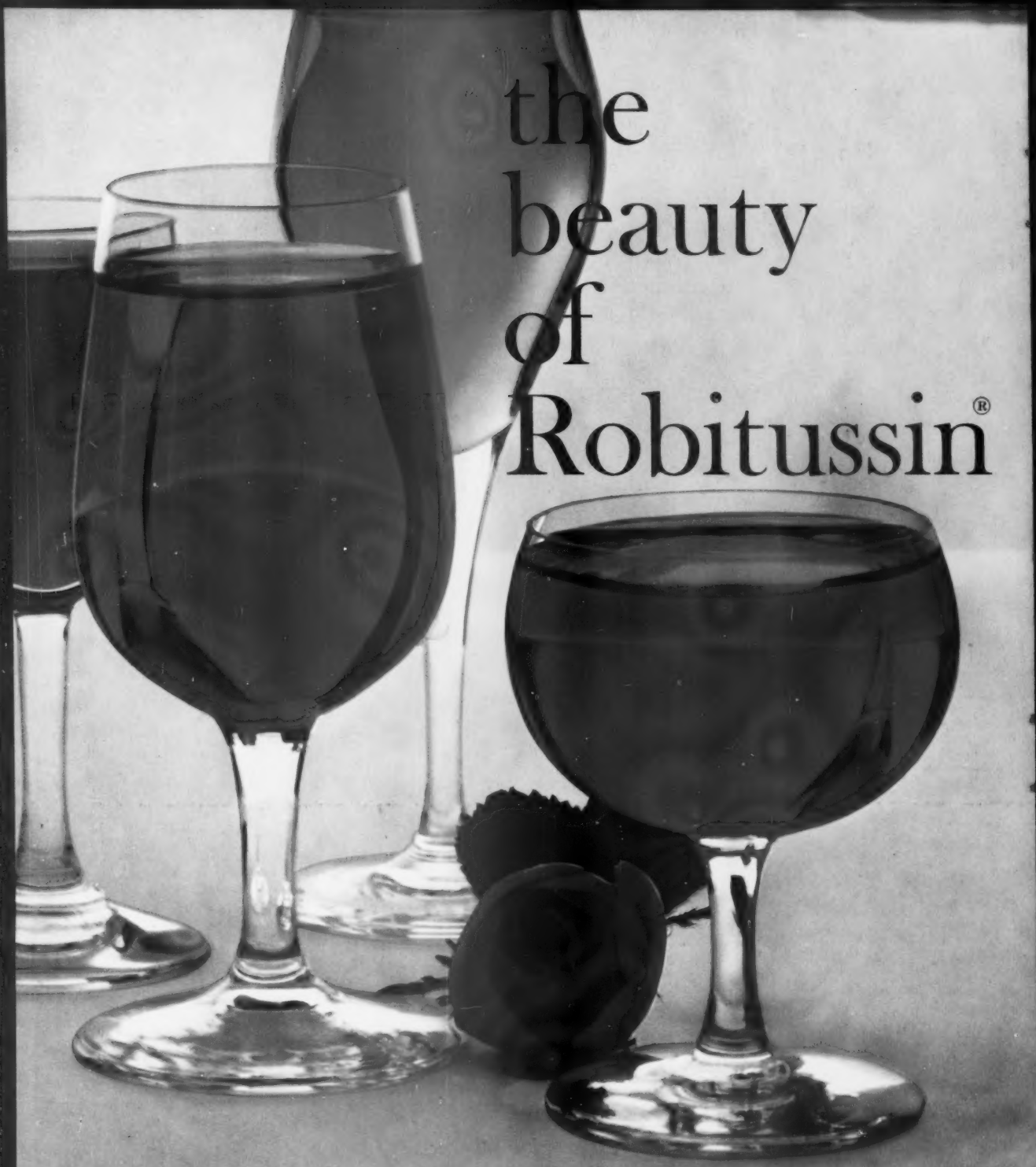

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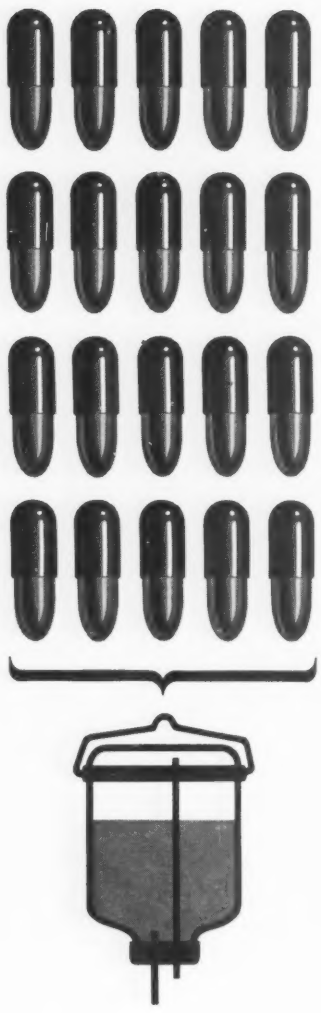

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The Virginia
MEDICAL
MONTHY

February, 1961
Vol. 88, No. 2
Whole No. 1305

Guest Editorial....

House Staff Recruiting and Hospital Standards

ALL APPROVED HOSPITALS must now submit to the results of the examination of the Educational Council for Foreign Medical Graduates in selecting foreign applicants for internships and residencies. This device purposes to raise standards of hospital care in this country by expelling and excluding from hospitals foreign medical graduates who fail to measure up to an arbitrary mark on an IBM type, one-day examination.

Whether this purpose will be achieved by this scheme remains to be seen. Meanwhile, certain obvious effects are being felt. The reduction of the number of foreign medical graduates, estimated at 40 to 50%, means that in many hospitals existing house staffs will be greatly depleted while others now having a house staff will have none. In such hospitals the obligation and opportunity to teach which is itself a stimulus to raise and maintain standards is lost; the presence of critical junior associates who regardless of their origin play the role of conscience in hospitals is gone; and many patient services formerly performed by professional hands are now necessarily relegated to technical assistants. Patently these immediate effects of the examination will not necessarily in themselves raise the standards of hospital care.

While admittedly many graduates of foreign medical schools are unfit to serve as interns or residents in our hospitals in this country, our own experience suggests that many men now serving with credit both to themselves and their countries will be dismissed and consequently deported in January, 1961, for having failed the examination this October. The full effect of hundreds of disgruntled young physicians returning to their homelands after distasteful experiences in United States hospitals may not be negligible and the number of foreign graduates who attempt to obtain positions here may well decline. This situation assumes paradoxical global

proportions in view of the great need for house officers here and the superabundance of medical graduates in foreign countries.

On the other hand, the net effect of the examination should be a healthy one. It is hoped that foreign universities will respond to the examination as a challenge to raise their standards of medical teaching. Improvements in the use of written and spoken English by many foreign graduates can be anticipated. Most important, United States hospitals must now look to their own house staff programs as they enter the inevitably increasing competition for obtaining interns and residents.

This brings us to the main point: a critical look at our own present methods of recruiting house staffs. What is it that attracts the medical graduate of our own schools into the post-graduate situation which he ultimately chooses? He knows that he is now in great demand. He will be "rushed" by hospitals as he has before by fraternities. In many instances he will be able to choose among several fawning services. What then are his criteria for selection of internship or residency?

His primary guide remains of course the educational quality of the internship and residency in itself. A good teaching program acquires a good reputation whether it be a medical center, a university hospital or a private or community hospital. Unfortunately, many hospitals with excellent teaching programs are understaffed. The results is hospital recruiting which offers inducements other than educational ones. As a consequence in choosing internship or residency positions, young physicians have established criteria other than those relative to learning and clinical experience.

One such secondary guide worthy of serious note is financial security. Here, government institutions including the Armed Forces, United States Public Health Service, Veterans Administration and various State institutions offer attractive intern and residency programs with high starting salaries, cheap insurance, retirement and other fringe benefits. These inducements appeal particularly to married students in financial need; in some instances such programs actually benefit students before graduation from medical school. We have recently had two competent house officers who after successfully entering practice chose employment in government service ostensibly for reasons of future security.

Another secondary criterion by which the young physician must deal with hospitals and their inducements relates not to education or security but to the immediate peripheral benefits of the programs: the gravy train, the hand-out, the *carpe diem* for the intern. Such seduction is simultaneously condemned and practiced by hospitals and their hirelings in

the business of acquiring house officers. The chronically acceptable practice of befriending students and presenting favorable facts of a hospital program becomes antiquated. Present recruiting procedures may now offer pyramidal salary boosts, country club membership, free tickets to festival football games, payola air travel to and from promotional get-togethers, more time off than time on, picnics, parties and balls of varying intensities and even a modicum of chauvinistic dealing on the part of department heads, administrators and physicians. Such promotional gimmicks are basically subsidization devices which appeal to anything but the best in young physicians.

In other states thoughtful measures are being taken to deal with the problem of house officer recruitment in an attempt to reconcile hospital demand to house officer supply. The most intriguing of these is the Temple University plan. It has become increasingly apparent that as medical center teaching evolves the intern in the university hospital tends to be squeezed between the rising competence of the senior student and the new-won authority of the junior resident. The Temple plan, as we understand it, makes fourth-year students full-fledged clerk-externs, releases interns to surrounding community hospitals and allows subsequent resumption of residency training. In the Michigan plan, university and non-university hospitals co-operate to share residency training needs for each other at various levels. In Jacksonville, hospitals have founded an educational program corporation which is intended to co-ordinate clinical material and organize a balanced program for the member hospitals. Here in Virginia with only 18 per cent of 150 approved non-university internships presently filled by the Matching plan, hospitals now faced with drastic curtailment of foreign house officers must take collective consideration of their plight. It is encouraging that efforts in this direction are already under way.

Clearly the standards of medical teaching are constantly improving both at pre- and post-graduate levels. However, the present mechanisms for resolving the dilemmas of supply and demand in the post-graduate phase are not necessarily those which assure improvement of the standard of medical care.

THOMAS N. P. JOHNS, M.D.

Problems Arising in the Medico-Legal Investigation of Deaths

ROBERT A. MORTON, M.D.
Norfolk, Virginia

It is well that all physicians understand precisely what the law requires of them in reporting deaths to the medical examiner. They should also know the medical examiner's duties and limitations in accepting cases and ordering autopsies.

SINCE THE ESTABLISHMENT of the Medical Examiner system in Virginia in 1946 it has become increasingly apparent that this is a successful, highly efficient means of medico-legal inquiry into certain types of deaths. The medical profession as well as law enforcement agencies, funeral directors and others are generally familiar with the operation of the System. Yet certain problems seem to recur chronically and the purpose of this paper is to explore and explain some of these problems as they relate to the medical profession. Frequently physicians do not realize their own responsibility when one of their patients dies and also may not be fully aware of the legal implications of a death occurring subsequent to an injury. It is apparent that most misunderstandings arise from the failure to understand the law from which the medical examiner derives his authority.

Chapter III, Title 19.1 of the Code of Virginia 1960, clearly outlines which deaths must be reported to medical examiners. These include the death of any person from violence, or suddenly when in apparent health, or when unattended by a physician, or in any suspicious, unusual or unnatural manner. The medical examiner is also to be notified in the case of a stillbirth occurring without medical attendance or a death occurring in a prison. The law also states that the notification is to be made by the *physician in attendance,* by any law enforcement officer, undertaker or *any other person* who has knowledge of such death. It is a medical examiner's duty to take charge of the dead body and make such inquiries and investigation as he deems neecssary to render an opinion as to the cause and manner of the death. He is empowered to order an autopsy only if the death is properly a medical examiner case and only if the establishment of the actual cause of death can be considered in the public interest. The legal interpretation of public interest does not include the satisfaction of academic curiosity or the establishment of accurate statistics. It does include the protection of the public health, detection of crime, identification of deceased bodies, differentiation between natural and violent deaths should such a controversy exist, settling questions of occupational hazards for the purpose of industrial compensation and the determination of the extent of injuries and recovery of weapons and missiles to aid in the prosecution of crime.

One of the most frequent problems relating to the medical profession is the failure to report deaths of individuals which actually represent violent or unnatural deaths in the eyes of the law, yet from a strictly medical standpoint may on the surface seem to be the result of natural processes. Generally the cases fall into one of two types of deaths. The first is a death that results from

aggravation by an injury of a pre-existing illness and the other, a death occurring at a time remote from the injury. Any death following an injury should be reported to a medical examiner. The medical examiner and physician must decide how much the injury actually contributed to the death. In general, if the injury *in any way* was a substantial contributing factor in the death, then under the law it is considered an unnatural or violent death even though the injury only aggravated the natural illness. From a legal standpoint, if the injury sets up a chain of events which leads to death regardless of the prior state of health of the individual or regardless of the time interval between injury and death, then the death is properly said to be due to that injury.

Case 1. In a recent case argued in a local court, an elderly male, who had suffered many years from bronchial asthma and had developed advanced emphysema and evidence of chronic respiratory failure, was struck by an automobile. The trauma resulted in two fractured ribs. The patient was hospitalized and approximately one week later succumbed to pneumonia. In court an argument developed around the fact that the patient could have been expected to develop pneumonia and die as a result of his pre-existing chronic pulmonary disease. Though this is true, the Judge upheld the opinion of the medical examiner that the rib fracture was a contributing factor to the pneumonia; therefore, this death was properly considered to be a violent death and should be categorized as due to a motor vehicle accident.

Recently two cases came under investigation in which the attending physicians had certified the cause of death because they felt it was due to natural causes, yet in both instances an injury had occurred which could be said to have led to the death. The certificates in these two cases were interpreted by the State Bureau of Vital Statistics to be improper death certificates since they had not been signed by a medical examiner.

Case 2. A 63 year old female had been confined to a nursing home because of advanced cerebral arteriosclerosis with marked mental changes. During the night of January 28, 1960, she fell out of bed suffering an injury to the right hip. The following morning she was seen by her attending physician at the nursing home who felt that she had suffered a fracture of the hip and transferred her to a local hospital. X-ray studies confirmed the fracture and on January 31 she had a hip-nailing procedure performed. At the time of admission she was in a confused state but this was a chronic condition and the fact that she remained confused post-operatively was attributed to her cerebral arteriosclerosis. On the third post-operative day she was noted to be semi-comatose and this condition progressed to complete coma. The attending physician and a consultant both felt that she had suffered cerebral thrombosis of an extensive nature related to a hypotensive episode during surgery but due to her extensive cerebral arteriosclerosis. She expired on the fifth post-operative day and the attending physician certified the cause of death as cerebral thrombosis due to cerebral arteriosclerosis. Mention was made on the death certificate that she had suffered a fractured hip due to a fall. When the death certificate arrived at the Bureau of Vital Statistics, the case was referred to the local medical examiner's office for investigation as a violent death.

Case 3. Another case, similar in nature, was that of an 89 year old female who during the night of February 21, 1960, fell at home. She was able to drag herself to a telephone and called her physician who admitted her to a local hospital on the morning of February 22nd. X-ray studies showed an intertrochanteric fracture of the right femur. This patient had a past history of arteriosclerotic heart disease and had been under treatment for approximately 15 years. At 11:00 a.m. on February 23rd she was taken to the operating room and had a hip-nailing procedure performed. Shortly after leaving the operating room, it was noted that she had extreme tachycardia and hypoten-

sion. An electrocardiogram was suggestive of acute myocardial infarction. She remained in a critical condition despite treatment and died at 1:50 p.m. on the day of operation. Her attending physician reasoned that the cause of death was an episode of acute coronary insufficiency due to arteriosclerotic heart disease which had been present for 15 years. Therefore he certified the cause of death but he mentioned on the death certificate that the patient had suffered a fall and fractured her hip. Again the death certificate was held up and returned to the local medical examiner's office for further investigation.

In both the above cases the attending physicians reasoned that death was due to a progression of a natural illness. However, both made mention of a fall producing an injury and in reviewing the cases with them, both agreed that the fracture and subsequent surgery initiated a chain of events culminating in death. Therefore, both deaths represented unnatural or violent deaths and should have been reported to a medical examiner. Both cases represent hip fractures in elderly individuals and it is frquently argued that these fractures can be due to osteoporosis and actually occur prior to an individual falling. It is generally accepted that this situation does occur and a fall may be the result of rather than the cause of the fracture. Should a physician have demonstrative evidence or proof that osteoporosis is present and in his opinion the fracture is due to osteoporosis and precedes a fall or traumatic episode, then he is well within his right to certify the cause of death as due to natural causes and list on the death certificate that the fracture is due to osteoporosis and is not traumatic. Such a case then would not be an unnatural death and would not properly be a medical examiner case. However, if there is doubt in his mind or if he feels that the fall precipitated the fracture and death subsequently occurs, such a case should be reported. A fracture is considered to be of traumatic origin unless it is due to some disease or disorder of the bone itself. Obviously, if this disease or disorder is not diagnosed and so stated on the death certificate then the physician has been remiss in his duties and the reviewing authorities would not have sufficient information to properly categorize the death.

Death at a time remote from the injury but still due to that injury is the other major type of unreported case. The time interval is of no significance if it can be said that the injury initiated a chain of events leading to death. Thus, if an individual is injured in an automobile accident and due to that injury becomes paraplegic, lives twenty years and then dies as a result of a urinary tract infection as a complication of his paralysis, it is still an accidental death and must be reported to a medical examiner for investigation and certification. It is not unusual for people to survive relatively long periods of time with injuries to their nervous system and yet die as a result of that injury.

Case 4. Another case recently reported to the local medical examiner's office from the Bureau of Vital Statistics involved a 32 year old male who was injured on the afternoon of May 8, 1959, when he was struck in the back by a log while he was at work engaged in logging operations. He suffered an immediate onset of paralysis of both lower extremities and was brought to a local hospital for further treatment. Studies at the hospital showed a fracture-dislocation of the spine at the level of T-12 and L-1 vertebra. He was operated on immediately after admission for a decompression of the spine; however, he showed no improvement in the paraplegia. He remained in the hospital for approximately eight weeks and was then taken to the operating room to have a spinal fusion performed. Shortly after being given a general anesthetic but before the actual start of the operation, the patient suffered a cardiac arrest. He underwent a manual cardiac massage with resumption of spontaneous heart activity and was returned to his room. He remained in a critical condition and expired about eight hours after the initial cardiac arrest. Here the attending physician felt

that the cause of death was cardiac arrest due to a general anesthetic and he certified the cause of death as such. However, he also mentioned on the death certificate that the patient had suffered a fracture of the spine due to an accidental injury. The certificate was returned for investigation by a medical examiner.

Upon investigation of this case, the attending physician felt that the time interval between the injury and subsequent death was sufficiently great to rule out the injury as being the primary factor in his death. From a strictly medical standpoint his death was unquestionably a result of cardiac arrest due to general anesthesia but from a legal standpoint the initial injury was what necessitated the general anesthesia, therefore, his death should have been classified as a violent death and a medical examiner notified at the time of the death. But, if the anesthesia had been necessitated by a natural illness (e.g. appendicitis) and the same result occurred, it would have been a natural death and the attending physician would have been correct in certifying the cause of death.

It becomes apparent that to properly report cases, physicians must keep *all* the facts in mind regarding a death. If violence or unnatural causes are present either as aggravating or precipitating factors the case must be reported to a medical examiner for investigation and only he can certify the cause of death. If a death which should be a medical examiner case goes unreported, then obviously the law has been violated. The responsibility for failure to report a case cannot legally be shifted to hospital personnel, undertakers, etc., as the law requires all persons having knowledge of the death to report it to the medical examiner of the city or county where the death occurred (NOT where the injury occurred). Should it later come to the attention of the medical examiner that a death has occurred and the body buried without proper inquiry, then he is obligated to notify the Commonwealth's attorney of such death, investigate the case as best he can and report his findings. The Commonwealth's attorney then, by law, must present the case to the Judge of the Corporation or Circuit Court for whatever action the judge deems proper. This may include the exhumation and autopsy of the decedent which would greatly increase the cost to the State and render the investigation much more difficult. To date, no physician in Virginia has been prosecuted for failure to notify a medical examiner of a medical examiner case. However, the law is clear and such action could be taken by a Commonwealth's attorney and no doubt would be if he felt that there was any intent to circumvent the law.

Another major problem facing medical examiners in dealing with the medical profession is that of being requested to authorize autopsies. Again, failure to understand the legal standpoint seems to cause the most friction in this matter. Is it not just as unreasonable to request a medical examiner to authorize an autopsy when he has no legal right to do so as it is to request a pathologist to perform an autopsy without permission of the next of kin? A medical examiner as an individual is just as responsible legally for his actions as anyone else. Two criteria must be satisfied before a medical examiner can legally and properly authorize an autopsy. First, the case must be under his jurisdiction and, second, the autopsy must be in the public interest.

It must be kept in mind that the State, through its medical examiners, is interested primarily in the manner of death rather than the exact medical cause. The manner of death here means that the death should be categorized as natural, accident, homicide or suicide. The exact cause from a legal standpoint, is of secondary importance. Thus, if an individual under attendance by a physician for an illness which could reasonably be expected to cause death, should die and there are no circumstances to suggest that the death is other than natural, the case is not necessarily reportable and if reported will be refused by the medical examiner. Frequently the attending physician feels that

if he professes ignorance of the cause of death, then the medical examiner will of necessity order an autopsy to determine the cause of death. This is fallacious reasoning. If a death clearly falls into one of the categories mentioned above, an autopsy would be considered superfluous from a legal standpoint, therefore, would not be in the public interest. However, should a controversy exist as to the manner of death (not the exact cause) then in all likelihood the medical examiner will order an autopsy.

Case 5. A 56 year old male was injured in an automobile accident on January 14, 1960, and admitted to a local hospital. Studies showed the presence of rib fractures on the left with a partial left pneumothorax. The patient also had a history suggestive of arteriosclerotic heart disease with an anginal syndrome. He seemed to respond well to initial treatment and his lung re-expanded with the use of a chest tube. However, he continued to complain of pain in his left chest and his electrocardiogram showed evidence of myocardial ischemia. His blood pressure fell to slightly hypotensive levels where it remained until about 7:00 p.m. January 17, when he suddenly went into shock and became comatose. In spite of emergency treatment, he died in approximately one hour. Here confusion existed as to whether the patient had suffered a myocardial infarction and succumbed or whether he had died from some unforeseen complication of his injury or from some unrecognized injury. A medical examiner authorized an autopsy on the basis that the manner of death was not clear. The autopsy disclosed an unrecognized rupture of the spleen and an essentially normal heart. Thus, the death was properly categorized as an accidental death.

It must be remembered that the surviving relatives of a deceased individual have certain rights in regards to the remains of the individual. They have the right to receive and bury those remains without undue interference. The medical examiner must respect those rights as well as anyone else. It is only in a situation where the interests of the State transcend the interests of the individual that a medical examiner can assert his authority and order an autopsy. On the other hand, medical examiners receive requests that autopsies be performed because the surviving relatives want it done. Here again, unless it is properly a medical examiner case, the medical examiner cannot legally enter into any such arrangement. In like manner, even if it were a medical examiner case that the surviving relatives wanted autopsied, unless the medical examiner felt that the public interest would be served by an autopsy, he would not be within his power to authorize the expenditure of State funds or the use of State facilities for such an autopsy. As a matter of information, most pathologists will agree to perform autopsies even on individuals who die outside their particular hospitals should they receive proper authorization from the family. Under these circumstances, the pathologist is entitled to a fee for his services since he is performing a service requested by the family. Even in clear cut medical examiner cases where the medical examiner does not authorize an autopsy, once he releases the body from his jurisdiction, then it may be autopsied if proper consent is obtained by the attending physician or pathologist. In this respect, attending physicians and house staffs of hospitals are encouraged to obtain permission for autopsy even in a medical examiner case. However, this must never be done until the medical examiner has made his investigation and released the body without ordering an autopsy. Where the family is approached prior to the medical examiner's investigation and decision and the family refuses permission, there may arise a suspicion in the family's mind that subsequent ordering of an autopsy by the medical examiner was done to circumvent their refusal.

As a matter of interest, there are a number of successful suits on the records against medical examiners or coroners for ordering autopsies where they had no clear legal right to do so. Most of these suits have occurred

in other states under a coroner system where the coroner can authorize autopsy only on the basis that a crime is suspected. However, the Virginia law uses "public interest" as a basis for authorizing autopsy rather than a mere suspicion of crime. To date, no successful suit has been entered against a Virginia medical examiner and this primarily is because the medical examiners have been well indoctrinated into what exactly constitutes their rights and duties.

Another major source of friction between the medical profession and medical examiners is the failure of physicians to understand that the physician last in attendance must certify the cause of death except in medical examiner cases (Chapter 32, paragraph 353.20, Code of Virginia 1960). Thus, if a medical examiner rules "not a medical examiner case" the undertaker must present the death certificate to the physician last in attendance for his certification and signature. The physician cannot shift this responsibility simply by pleading ignorance of the exact cause of death. The law does not require that the certification be accurate beyond all doubt, but does expect a physician to arrive at the probable and most reasonable cause of death based on his knowledge of the patient, the patient's illness and his store of general medical information. A medical examiner is not within his right to accept a case because the attending physician does not wish to sign a death certificate. The case must properly be a medical examiner case and then only a medical examiner can sign a valid certificate. Physicians who pursue a course of obstinate refusal to sign a death certificate on one of their own patients are obstructing the usual orderly process of funeral and burial, creating difficulties for undertakers and the surviving members of the family and are breaking the law. The Bureau of Vital Statistics can issue a complaint against the offending physician resulting in his criminal prosecution.

In summary, the major problems that the medical examiner system encounters with the medical profession are those of failure to report cases legally considered unnatural deaths but, which medically appear to be an aggravation of pre-existing illnesses; failure to report cases in which death occurs at a time remote from an injury and attempts to have a medical examiner authorize autopsies in order to establish an exact cause of death. If the physician will report all cases in which death occurs subsequent to an injury the first two of these problems will be eliminated. If he will make some attempt to understand the legal basis and implications of ordering autopsies, he will well understand why a medical examiner will not authorize autopsies in cases where no controversy exists as to the manner of death. If he will understand that the law requires the last physician in attendance to certify the cause of death in the case of a natural illness and that this requirement cannot be shifted to a medical examiner by pleading ignorance of the exact cause of death, much friction will be removed. He should also understand that should a death be properly a medical examiner case, then only a medical examiner can sign the death certificate, and it is only this certificate which has any validity in a wide variety of legal implications.

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Acceleration Therapy of Recurrent Depressions

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Toxicity has not occurred when utilizing two antidepressant agents. However, close control of the medication is the safest factor in preventing undesirable side effects.

FOR MORE THAN TWO YEARS psychiatrists have been fortunate in having at their disposal a number of excellent and well proven antidepressant agents. The first was Marsilid in 1957, then Catron in 1958. During 1959, Nardil, Marplan, Niamid, and Tofranil made their appearance on the scene. By now, many hundreds of thousands of cases have been reported showing substantial benefits following the use of these antidepressant agents. Although they have most assuredly assumed a major role in the treatment of the depressed patient, earlier enthusiasm for these drugs must be looked upon with some reserve as they have not and probably will not in the immediate future, at least, replace electroshock therapy, the "Prima Donna" of all psychiatric therapies for the depressed patient.

Many of the enthusiastic reports have included the excellent response to antidepressants in patients who have previously failed to improve following electroshock therapy. Many of us have long recognized a certain number of these "hard core" patients who resisted electroshock therapy and have proven disappointing until antidepressant agents provided the key to recovery.

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The nature of depressed reactions in many cases tends to be a recurring one and we have been faced with the return of patients who are suffering a recurrence of their depression which had been successfully treated by an antidepressant agent a year or two before. By the time the patient returns again he has invariably resorted to the antidepressant of choice which had previously been prescribed with such success. He has given it a fair trial, being fully aware that the drug does not work immediately and that a lag period of two to three weeks is often required before therapeutic benefits are seen. When this unimproved patient comes to the office, one is sometimes at a loss as to what course of treatment to follow next.

Invariably the patient feels that electroshock is still a major procedure and he will usually protest against its use. Having seen the disappointing results that follow when one simply prescribes the same antidepressant in the same amount as was used formerly, then failing to show substantial improvement, we decided to include in the treatment an additional "psychic energizer" to, perhaps, facilitate the recovery of the patient.

A number of patients with a history of cyclic or periodically recurring depressions have been satisfactorily controlled by the combined use of two antidepressant agents. To obtain such control usually takes from 4 to 21 days. Following this, most of these individuals remained healthy with a daily maintenance dose of 5 to 10 mg. of Nardil per day. In both groups the rate of recurrence was decreased significantly. The two patients described below are representative of a group of about 20 such cases.

Case #1, G. L. Diagnosis: Recurrent Depression: This patient had failed to recover after a series of 28 electroshock treat-

ments in the winter of 1958. Later Nardil, 15 mg. t.i.d. produced a dramatic improvement in about one month and the patient enjoyed her best health in years. Her depression, however, recurred late in 1959 but Nardil, given as before, was not effective. Catron 6 mgm b.i.d. for five days was then prescribed to supplement the Nardil. Within five days the patient showed considerable improvement. Therapy continued at the level of 6 mg. Catron daily and Nardil, 15 mg. t.i.d. Within three weeks very substantial improvement resulted. Patient is now being satisfactorily maintained on Nardil alone, 5 mg. b.i.d.

Case #2, B. C. This patient had a recurrent psychotic depressed reaction which was successfully treated four years ago with electroshock therapy. "Her condition recurred severely in 1959 and the patient refused to be hospitalized and refused to submit to electroshock." She was given Nardil six weeks; there was still room for further improvement. Catron, 6 mg. b.i.d., was added to this patient's medication and within a week, noticeable improvement had materialized. She remained on this therapy for an additional two weeks and is now being maintained in excellent condition on Nardil 5 mg. b.i.d.

Name	Age	Diagnosis	Prev. Treatment	Synergistic Agent	Onset of Improvement Noted	Result	Maint. Schedule
G.L.	43	PPDR	t.i.d. Nardil 15 mgm.	Catron 6 mg. t.i.d.	4 days	Excell.	Nardil 5 mg. daily
M.W.	74	IDR	t.i.d. Niamid 25 mgm.	Catron 6 mg. b.i.d.	14 days	Excell.	Niamid Catron
D.H.	48	DR	t.i.d. Tofranil 15 mg.	Catron 6 mg. b.i.d.	8 days	Excell.	Catron 1 daily
E.D.	72	IDR	t.i.d. Tofranil 15 mg.	Catron 6 mg. daily	14 days	Excell.	Tofranil 15 mg. daily
D.R.	56	DR	d.i.d. Marplan 10 mg.	Catron 6 mg. b.i.d.	7 days	Excell.	10 mg. daily Marplan
R.G.	32	PPDR	t.i.d. Nardil 15 mg.	Catron 6 mg. b.i.d.	4 days	Good	Catron 6 mg. Nardil 5 mg.
H.S.	45	RD	q.i.d. Niamid 25 mgm.	Catron 6 mg. b.i.d.	5 days	Good	None
L.C.	68	IPDR	t.i.d. Nardil 15 mgm.	Catron 6 mg. b.i.d.	12–14 days	Excell.	Catron 6 mg. Nardil 5 mg.
H.F.	40	RD	t.i.d. Nardil 15 mgm.	Catron 6 mgm. b.i.d.	7 days	Good	Catron 6 mgm. Nardil 15 mgm.
O.G.	50	RD	t.i.d. Nardil 15 mgm.	b.i.d. Catron 6 mgm.	10 days	Excell.	None
L.C.	60	IDR	Marplan 10 mgm. t.i.d.	Catron 6 mg. daily	14 days	Excell.	Marplan 10 mg. b.i.d.
S.K.	38	RD	Niamid 25 mgm. q.i.d.	Catron 6 mg. b.i.d.	12 days	Good	Niamid b.i.d. Catron 6 mgm.
N.B.	26	RD	Niamid 25 mgm. q.i.d.	Catron 6 mgm. t.i.d.	5 days	Excell.	Catron 6 mgm.
J.A.	66	IPDR	Marplan 10 mg. t.i.d.	Catron 6 mgm. b.i.d.	14 days	Good	Marplan 10 mg. b.i.d.

SCALE:
PPDR—Postpartum depressed reaction
IDR—Involutional depressed reaction
DR—Depressed reaction
RD—Reactive depression
IPDR—Involutional psychotic depressed reaction

Summary

From our experience in treating many hundreds of patients with various techniques, it is obvious that we will continue to see recurrence of the former depressive state. Not always does simple restitution of previous treatment prove as effective as it did in the first instance. It often only discourages the patient from continuing on the same therapy if noticeable improvement is not obtained; therefore a more encompassing program should be considered.

Our experience has been contrary to that of Ayd.[1] At first we were concerned that by utilizing two antidepressant agents, toxicity might be produced whereas none had appeared when the agents were used singly. This toxicity has not occurred and we recognize that close control of the medication is the safest factor in preventing undesirable side effects.

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Millions Spent for Cold Remedies

Cold remedies relieve Americans of millions of dollars each year but they do not cure or prevent colds, according to a publication of the American Medical Association.

"No cold remedy can do anything more than provide temporary relief of certain cold symptoms," an article in the January Today's Health said.

"Many do not even relieve symptoms effectively. Hence, they do little more than subtract from your pocketbook and add to the feeling that you've done something."

There is no known drug which will cure a cold, one reason being that not all the viruses which cause the ailment have been found, the article said. Another reason is that no one has discovered an antibiotic that kills viruses as they do bacteria.

"In the best scientific opinion today, a cold is a nose and throat infection that lasts about a week and is caused by an unknown number of viruses. To date, at least six of these viruses have been isolated." When all the viruses have been found it may be possible to produce a preventive cold vaccine.

"The cold remedy racket owes much of its success to the fact that the cold is a self-limiting disease." In other words, the symptoms will disappear naturally in a matter of days whether medicine is taken or not.

Nevertheless, Americans spent $309,350,000 for package cold and cough medicine off the drugstore shelf in 1959, according to one estimate.

The article offered the following advice on what to do when you catch cold:

—Stay at home and take other precautions to prevent spreading the virus to others.

—Go to bed and rest, keep covered, and avoid drafts.

—Eat an adequate, well-rounded diet and drink plenty of fluids.

—Blow your nose gently when necessary.

—Use steam from a kettle or vaporizer to relieve nasal congestion.

—If your cold persists for more than a week, if you have more than one degree of fever for more than a day, or if you get more than three or four colds a year, see your physician.

The article was written by Theodore Berland.

Diabetes Mellitus

Re-Examination of the Diagnostic Symptoms, Signs and Tests

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It is well to review the diagnostic tests, symptoms and signs of this widespread and most insidious disease.

IN THE PAST SEVERAL YEARS basic science research and clinical observations have determined the cause of certain metabolic and endocrine ailments heretofore listed as "diseases of unknown etiology". Further it has stressed the existence of endocrine and metabolic diseases in prodromal and mild states; thus pointing out the fallacy of depending on the "typical text book picture" before making a diagnosis. Not the least of the metabolic diseases is diabetes mellitus. It has been estimated that there are approximately 2,900,000 diabetics in the United States and about 1,400,000 are as yet undiagnosed. The latter are made up of persons never examined and patients missed by physicians. The enormity of the diabetic problem however is easily verified in any physician's office or any hospital clinic. The problem of diagnosing these diabetics when they present with the typical complaints of polyphagia, polyuria, polydipsia, weakness and weight loss in association with sugar in the urine is not the difficulty confronting the physician. It is the atypical diabetic, with few or none of the usual signs and symptoms, that presents the diagnostic challenge. Only when the clinician develops a "High Index of Suspicion" and makes use of the available clinical information and laboratory tests, will he "pick up" these difficult to diagnose diabetics. It is the aim of this paper, therefore, to attempt to stimulate somewhat this index of suspicion and to review the more recent clinical observations and the laboratory tests pertinent to the diagnosis of diabetes mellitus.

As stated by Dr. Best,[1] whatever the basic defect of diabetes, the result is in general metabolic dysfunction affecting protein, fat and minerals as well as carbohydrate metabolism. For diagnostic test purposes the glucose intolerance is most important, although the clinical features may be due to the other dysfunctions. The former is due either to a complete lack of secretion of insulin by the pancreatic islet cells, which gives rise to the unstable or juvenile type of diabetes; or due to a relative lack of insulin (when there is usually 50% or more of the normal amount of insulin existent in the pancreas and blood), resulting in the stable or adult type of diabetes. A third type of diabetic which is being described more and more in literature is known variously as the prodromal, sub-threshold or pre-diabetic. The pre-diabetic is as the terminology suggests, a diabetic who has not as yet manifested overt diabetes, but who generally will show a glucose intolerance if stress factors are applied. For diagnostic purposes a term "Dif-Diabetic" has been coined as an abbreviation of the "difficult to diagnose diabetic". This group, as will be seen, is not entirely confined to the pre-diabetic or mild type but includes even rather severe glucose intolerants.

Review of Laboratory Tests

The *routine urinalysis* is the most widely used test for mass surveys and office screening of the population for diabetes. There are

available several methods for testing for urine sugar, all apparently equally sensitive for diagnostic purposes[2,3] (Benedict's solution, Clinitest, Tes-Tape, Clinistix and other glucose-exodizing papers). However the urine test is the least valuable procedure even when done at the recommended time of two to three hours after meals, because it is only significant if positive. A patient may have a high renal threshold or have a mild diabetes which is in balance at the time of testing and therefore present a negative urinalysis. The case of a 68-year-old white male points out the former of these two problems very vividly. This patient developed symptoms of cholecystitis with lithiasis in 1958, which was proven by x-ray. He refused surgery and in the course of the next twelve months went downhill with the complaints of weakness, weight loss, peripheral neuropathy and cardio-vascular symptoms and signs. Urinalysis done on repeated occasions were all negative for sugar and the patient was finally admitted to the hospital in 1959 for surgery with the diagnosis of probable biliary or pancreatic carcinoma to account for the downhill course. On admission to the hospital the routine blood sugar was 230 mgm.% and the urine still failed to show sugar. Surgery revealed cholecystitis with lithiasis and it was apparent that his downhill course was due to the undiagnosed diabetes.

The *routine blood sugar* should be done as readily as we do the routine hematocrit, urea, etc. The value of this latter is exemplified in the case of a 60-year-white male, a known Hodgkins disease, who was admitted for retreatment because of weight loss, weakness, scattered shoddy lymph nodes and a solitary rib lesion. A routine fasting blood sugar was 131 mgm.% and a subsequent glucose tolerance test revealed that at least part of his disability at this time was due to diabetes. (see Fig. 1). On the other hand the routine fasting blood sugar has its limitations, because the mild to moderate glucose intolerant may manifest a normal fasting blood sugar. An example of this is the Case of S. D., who was admitted to the City Hospital in Binghampton, N. Y., in 1949 with an osteomyelitis of the distal phalanx of the

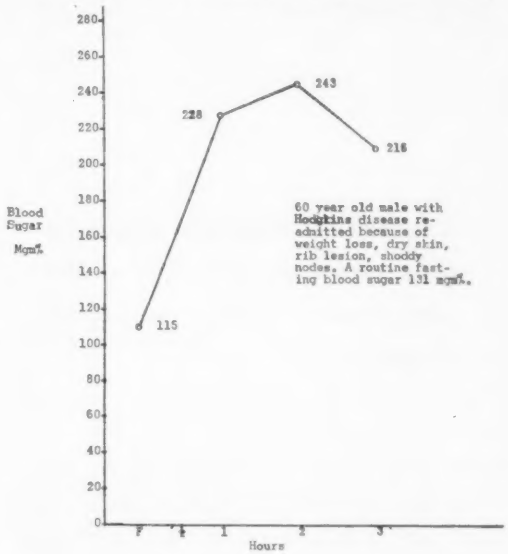

Fig. 1

right thumb. This patient had a history of having had a trace of urine sugar at one time. His fasting blood sugar was normal but it was decided to do a glucose tolerance test because of the history of the urine sugar. As one can see in Fig. 2, the fasting sugar was

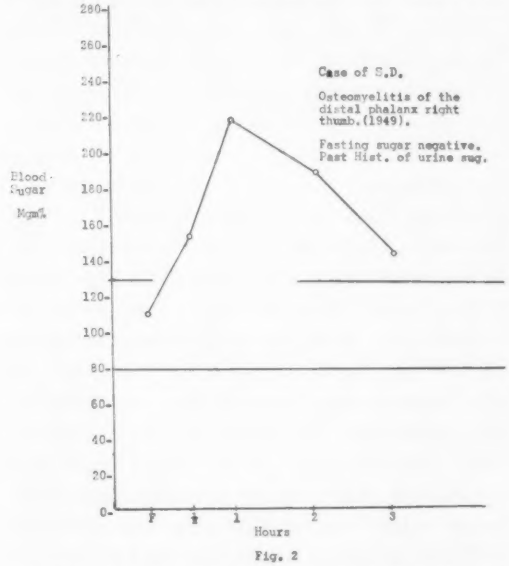

Fig. 2

again normal, but the curve is definitely indicative of diabetes. Another point to re-

member is that a positive urine sugar followed by a normal blood sugar, even if collected at the same time, does not mean "no diabetes". The bladder may contain sugar from a previous renal spill when the blood sugar was elevated. A much better routine screening test is the two hour post-prandial blood sugar. However one should be careful that the patient does receive an adequate amount of carbohydrate with the meal, even if he has to supplement with 50 to 100 grams of oral glucose. Otherwise the patient may not eat enough to give a test much better than the fasting blood sugar. By supplementing the meal with additional glucose one is actually doing a modified glucose tolerance test. Allen, et al[4] have devised a modified screening test by giving the patient two bars of coconut penuche fudge containing approximately 100 grams of sucrose. They state that there has been better patient acceptance and less nausea as is experienced with the oral intake of 100 grams of glucose.

An interesting screening test for blood sugar is the Dextro-test Kit manufactured by the Ames Company. My experience with about 170 tests done on office patients has been that it is a good economical means of following the control of the diabetic, but as a screening diagnostic test for diabetes it gives a high percentage of false positives. This latter is due principally to the difficulty in grading the test between 100 mgm.% and 150 mgm.% blood sugar.

A few words about the methods for determining blood sugar in the laboratory. It is my understanding that most laboratories use the Folin-Wu technique, but some use the "true sugar" Somogyi method. In the Folin-Wu determination certain reducing substances other than glucose are measured, so that the actual reading is 20 to 30 mgm.% higher than the true blood sugar level. The Somogyi method removes these other reducing substances and therefore the final blood sugar readings will be lower than with the former method. Thus the normal blood sugar ranges from 65 to 110 mgm.% Somogyi and 80-120 mgm.% Folin-Wu. Therefore in evaluating the blood sugar found, whether fasting or post-prandial, if your laboratory uses the Folin-Wu method then any reading 130 mgm.% or above is suspicious to diagnostic depending on the level, and in the case of the Somogyi method a blood sugar 120 mgm.% or above has the same significance.

The *Standard Glucose Tolerance Test* is indicated if the patient fails to reveal definite sugar intolerance by the fasting or two hour post-prandial blood sugar. It has been a standard procedure to prepare the patient with at least 300 grams of carbohydrate per day in their diet for as much as three days prior to the tolerance test. This is thought to eliminate the possibility of false positives by utilizing the islet cell stimulating effect of glucose to insure maximum insulin output by the pancreas. However some recent work reported by Wilkerson, et al[5] suggests that the necessity for the routine preparation has been exaggerated. They fed pre-test patients diets varying from 50 grams to 300 grams of carbohydrate daily for from three to seven days. They found no false positive diagnosis of diabetes in a group of 57 non-diabetic men and women. On the other hand it has been generally recognized that repeated glucose tolerance tests on the same patient creates an increasing tolerance to glucose. There has been some question as to whether the oral glucose tolerance test or the intravenous test is most accurate. One study indicated that the oral tolerance test gives abnormal curves in 15% more individuals than the estimated prevalence of the disease. However the oral test seems to be practical and diagnostic in the individual patient who has been properly prepared and free of malabsorption dysfunction. Generally 50 to 100 grams of glucose are given by the oral route and 25 to 50 grams intravenously.

Regardless of the route of administration the evaluation of the glucose tolerance curve is the same. It can be divided into three phases. The *initial or fasting glucose level* in the normal, mild or even moderately severe diabetic will usually vary between 70

and 120 mgm.% (Folin-Wu). If it is above 130 mgm.% it is diagnostic in itself of diabetes, providing one rules out advanced nephritis, functional (emotional) hyperglycemia, hyperthyroidism, hyperadrenalism and other endocrine ailments. The *second phase* is the peak of the curve occurring in one-half to one hour. The average normal limit is 180 mgm.%, but it varies with age. In the young most normal peaks are 160 mgm.% or lower and in the aged may reach 190 mgm.% normally. It is during this phase that an abnormally high blood glucose can occur in the patient with hepatic disease or functional hypoglycemia (alimentary hypoglycemia). This latter fact is what makes *phase three* or the descending limb of the curve the most important in the diagnosis of diabetes. For unlike the fall of the blood sugar to normal or abnormally low levels in the hepatic or hypoglycemic syndromes the blood sugar will remain elevated above 130 mgm.% in two hour and three hour specimen. However it may return to normal range in three hours in the mild diabetic. (Fig. 3)

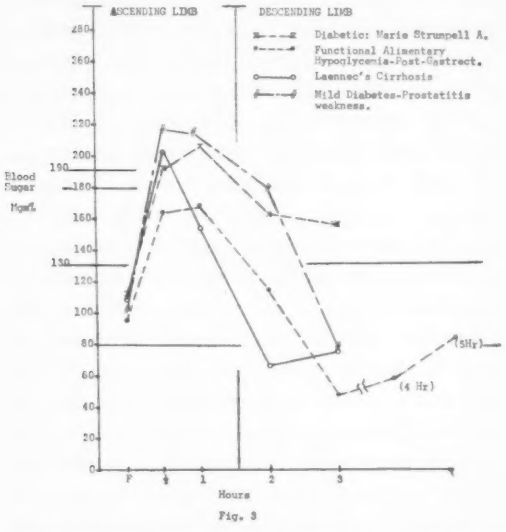

Fig. 3

The *Cortisone-Glucose Tolerance Test*, as devised by Conn and his associates[6] is based on the observations that cortisone reduces insulin sensitivity and thus glucose tolerance. (The probable mechanism for the appearance of diabetes during pregnancy.) They have calculated that giving patients weighing less than 160 pounds 50 mgms. of cortisone acetate eight and a half hours and again two hours prior to a standard glucose tolerance test, will cause an abnormal tolerance curve in patients with diabetes or diabetic tendencies (pre-diabetic), but will not affect patients with normal glucose tolerance. (Patients weighing more than 160 pounds are given 62.5 mgm. of cortisone acetate.) This test is used principally to determine diabetic potentiality, and as will be noted has been extremely important in adding to our knowledge of the pre-diabetic state. The results of this test are interpreted the same as outlined above for the standard tolerance test.

The *Intravenous Tolbutamide* (*Orinase*) *Response Test* is thought by some to be the best method of determining the mild diabetic. The patient again is prepared with three days of high carbohydrate intake. A fasting blood sugar is drawn in the morning, then one gram of Orinase diluted in 11 c.c. of distilled water is given intravenously in two minutes time. Blood glucose determinations are then done at 20 and 30 minute intervals. The results indicate that in the diabetic the 20 minute specimen will remain at a level of 84% or more of the fasting specimen, whereas the non-diabetic will show a drop in blood sugar of 80% or more of the fasting level. Further evidence of glucose intolerance is noted when the 30 minute specimen fails to decrease below 77% of the fasting level. (Fig. 4)

This test can be preceded by a stress dose of cortisone, as outlined above, and it too becomes a very sensitive test for study of the pre-diabetic patient.

The *Six-minute Insulin Tolerance Test** measures the patient's sensitivity of resistance to insulin and therefore is useful in separat-

* This test described by Sir Harold P. Himsworth of London and procedured in this country by Dr. George E. Anderson of State University College of Medicine, New York City.

ing the juvenile or unstable from the adult or stable type of diabetic. An interesting finding with the test was that the obese diabetic manifesting insulin resistance became normally sensitive to insulin with weight loss.

The procedure of testing is to determine the fasting blood sugar and then inject three

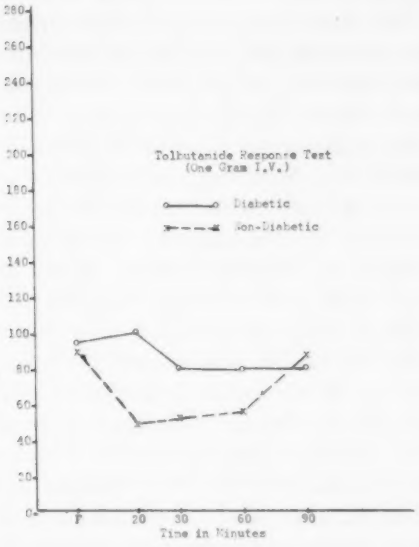

Fig. 4

units of glucogon free insulin intravenously. Blood samples are then examined for glucose level at two, four, and six minute intervals. *Normally* the blood sugar falls from six to 26% of the fasting level within six minutes. There may be an initial rise, but this is followed by a drop. The *insulin-resistant* patient has a flat curve or may even show a slight rise during the six minutes. The *juvenile diabetic or insulin sensitive* patient shows a precipitous fall of blood sugar, usually within two minutes.

Review of the Clinical Observations Pertinent to the Diagnosis of Diabetes Mellitus

As stated previously, if the practicing physician depends on typical clinical symptoms and findings of urine sugar or even hyperglycemia on a single blood specimen he is going to fail to diagnose a great number of mild, moderately severe glucose intolerances and pre-diabetics. An awareness of certain clinical leads and a "High Index of Suspicion" along with the systematic employment of the tests just reviewed are necessary. In the following paragraphs some of these diagnostic clinical features will be discussed.

The *typical, although nonspecific, complaints* are those of loss of strength in about 64%; polyuria 73%; polydipsia 67% and polyphagia in about the same percentage. The latter is pointed out only to indicate that a good number of diabetics will have few if any of these symptoms. On the other hand, it has been noted in the past by diabetologists and re-emphasized by the employment of the cortisone stress tolerance tests, that the above symptoms and other genito-urinary complaints such as burning, urgency and itching, may manifest before the actual onset of hyperglycemia, at least the glucose intolerance may be so mild that it is easily overlooked. An example of this is the case of a 48-year-old white female who was admitted to the Shenandoah Hospital in Roanoke, Virginia, in 1957. Her chief complaints were those of weakness, urinary frequency, burning, itching and vague abdominal pains. The urological work-up indicated urethritis and cystitis. A general medical work-up was indeterminate and such things as hypothyroidism, diabetes, hyperparathyroidism, gastrointestinal disease and others were apparently ruled out. (Among others the laboratory tests showed PBI: 6:1; BMR: plus 5; Cholesterol: 142 mgm.%; blood sugar: 124 mgm.% fasting and all urines negative for sugar.) The patient was treated for probable menopausal syndrome, but her symptoms of weakness and fatigue persisted and although the urinary symptoms had abated, they recurred. She was re-admitted to the hospital in 1959 and a fasting blood sugar was 132 mgm.% (urine negative) and a three hour post-prandial blood sugar was 169 mgm.% This patient may represent a pre-diabetic problem, but probably was a mild diabetic that was missed in 1957.

Family History: The inheritability of diabetes has been a question of much debate and study. More recently it has been generally assumed that it is transmitted by the Mendelian recessive characteristic. The diagnostic importance of a positive family history, especially if on both sides of the family, cannot be stressed too much. It has been pointed out by many, including Fajans[7] and Conn[6] that the standard glucose tolerance test will be abnormal in 18% of the apparently healthy relatives of known diabetics in contrast to 1 or 2% in a control group. If the cortisone stress test is applied, the figure jumps to 25% in the relatives of known diabetics, in contrast to 2-3% of the controls. Further application of these tests to apparently healthy *obese* relatives of known diabetics indicates that the overweight patient in our office with a family history of diabetes, is a *must reduce candidate*. This latter takes into consideration the fact that 80 odd per cent of all diabetics are overweight. One must remember that the onset of diabetes cannot be predicted by date or age, and some member of the family may develope overt diabetes between office examinations. Therefore the question should be repeated of the same patient.

Dermatological Signs: Certain skin conditions should put us on our guard. We are all familiar with the fact that furunculosis and fungous infections of the skin and the latter particularly of the nails are frequent complications of uncontrolled diabetes. This is also true of monilia infections of the mucous membranes of the vagina. In fact, pruritus vulvae is so often associated that it demands the exclusion of diabetes. There are certain skin lesions less frequently encountered such as *Necrobiosis Lipoidica Diabeticorum, Xanthoma Diabeticorum and Xanthelasmia.*

Necrobiosis L. D. causes a reddish papular lesion about 1-3 mm in diameter. It occurs singly or may be multiple, growing slowly and ulcerating. It is a rare lesion with no known cause. *Xanthoma* is the most rare of these lesions, consisting of a yellowish plaque appearing chiefly on the elbows, knees and buttocks, but also other skin areas and buccal mucosa. It is apparently directly or indirectly influenced by the blood cholesterol level because it disappears when diabetes and the latter are controlled. *Xanthelasmia* is a more common finding consisting of a yellow plaque on the eyelid. It is found in diabetics and non-diabetics. Vacca, et al[8] recently reported 51 consecutive patients with this lesion, of which they reported 35 patients who were evaluated for diabetes. Twelve of the 35 or about 35% had diabetes.

Delayed healing of surgical wounds and chronic infections, regardless of the fact that it may be explained on another basis, should also bring up the question of the presence of diabetes. A good example of this is the case of a 61-year-old white female admitted to the hospital with weakness, weight loss, persistent cough productive of purulent sputa and fever, and a vague history of thyroid disease. She had a goitre, emphysema, bronchiectasis and pneumonia in both lower lobes. Sputa examination ruled out tuberculosis, fungous infection and demonstrated pneumococci and streptococci. The RAI and PBI were normal. The blood cholesterol was elevated and a fasting sugar was normal. The glucose tolerance test however indicated diabetes. (Fig. 5) So even though this pa-

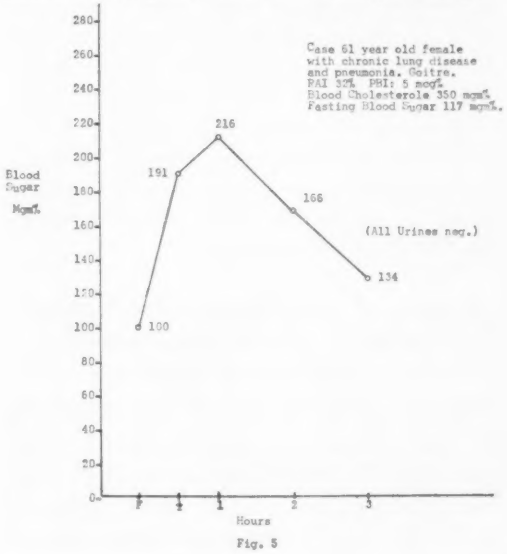

Fig. 5

tient's chronic lung changes were enough to account for her persistent pulmonary infection, she was found to have another causative factor.

Hypoglycemic episodes are not an infrequent clinical syndrome and are usually considered to be secondary to hyperinsulinism due to pancreatic adenomas, functional or alimentary hypoglycemia, hypoadrenalism, hypothyroidism, cigarette smoking and other causes. It has been recognized, however, that hypoglycemia may be part of the early symptomatology of sporadically compensated and uncompensated diabetes. *In fact this may be the chief symptomatology of the patient.* The basis for this seems to be the glucose stimulation of the pancreatic islets to produce insulin during a hyperglycemic state, thus leading to an excess of insulin. Later the pancreas loses this ability to be stimulated and the true diabetic picture emerges. This symptom may exist for periods of up to six years prior to the clinical recognition of diabetes.[9] The case of a 28-year-old white female represents such a problem. She gave a history of weakness, fatigue, weight loss, large appetite and clinical hypoglycemic episodes. Her mother's sister had diabetes. A urine sugar was negative and a three hour PC blood sugar was 92 mgms.% A glucose tolerance test indicated mild diabetes, with the blood sugar level sustained at 200 mgms. % in two hours falling to normal at three hours. Since the hypoglycemic symptoms depend more on the rate of fall of the blood sugar than the actual hypoglycemic range, a fall in one hour from 220 mgms.% to 105 mgms.% possibly could account for the headache, lightheadedness, nausea and other symptoms the patient had clinically and during the tolerance test. (Fig. 6)

"Benign Glycosuria": This term may well have a place in medical terminology, but if so, it surely is an exclusion diagnosis. The minutest trace of sugar in the urine should be looked upon with suspicion and a wholehearted effort made to rule out diabetes. This latter effort should go far beyond that of a repeat urine or a solitary blood sugar which are reported as normal. A glucose tolerance test is surely indicated if one fails to obtain a positive report from the blood sugar taken two hours post-prandially. As has been pointed out repeatedly in the mild and even moderately severe diabetic, one normal blood sugar does not rule out diabetes. Granted that one may find that the urine reducing substances is one of the non-glucose mellituria or possibly one of the non-sugar reducing chemicals such as salicylates, menthol, chloral hydrate, aminopyrine or even homogentistic acid found in alkaptomuria. (Remember, however, the glucose-oxidizers such as Tes-Tape, Clinistix, react only with glucose.) Also it is recognized that the renal threshold during pregnancy may be lower and one may find sugar in the urine with

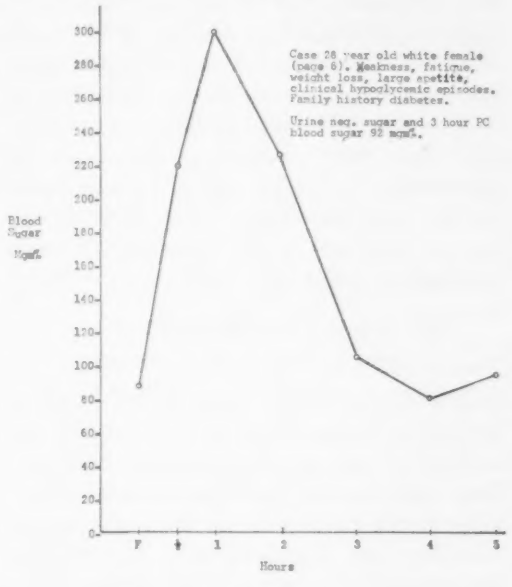

Fig. 6

renal ailments such as Fanconi's syndrome or renal tubular acidosis. However, all this does not obviate the fact that it may be diabetes. This is true also of the tendency to discount urine sugar spilled during a therapeutic intravenous of glucose. The possibility of a transient hyperglycemia because of starvation, hepatic disease or other causes is to be considered; however one must also consider the fact that the patient is receiving 50

grams of glucose with every 1000 cc of 5% glucose solution, and thus a glucose tolerance test. The case of a 59-year-old white male admitted to the Roanoke Memorial Hospital in 1959 is only one example of many such cases. Because of abdominal symptomatology associated with nausea and vomiting he was given an intravenous of 5% glucose in saline the night of admission. Urine collected that evening showed a two-plus sugar. A repeat urine the next day was negative, but a blood sugar done on the second day (about 24 hours after the I.V.) was 158 mgms.%.

In a series of 1140 cases reported by Pomeranze,[9] 96 had been told that they had insignificant urine sugar sometime prior to the development of full blown diabetes. The case of a 56-year-old white male is interesting, but perhaps not unusual. He was examined for life insurance in 1954, at which time sugar was noted in one urine specimen. He was sent home to return the next day, and he was told that his urine was clear at that time, and apparently obtained the insurance and a clean bill of health. His mother was a severe diabetic and in 1956 this patient was seen with a moderately severe uncontrolled diabetes.

Diagnostic Significance of the Complications of Diabetes

The clinical observations in the past and present in conjunction with the availability and use of the Cortisone-glucose tolerance test, Tolbutamide Response test and others, have focused attention on the fact that the complications of diabetes may exist prior to the onset of overt glucose intolerance or the diabetes may be so mild that it may escape detection. Dr. Freedman[10] sums this up very well with the following excerpt from his paper. "Renal failure with peripheral neuropathy and retinitis should arouse suspicion of diabetes even if no hyperglycemia or glycosuria is noted. The carbohydrate defect may be so slight that diagnosis can be made only on the basis of a glucose tolerance test. The patient may report having had symptoms of diabetes in the past. Destruction of the anterior pituitary or the adrenal glands or weight loss in a previously obese individual may ameliorate diabetes. Liver or thyroid disease may also influence carbohydrate metabolism." This latter is illustrated by the case of a 51-year-old white female who was treated for diabetes in 1937 by insulin and diet. She stated that in a few years she was "cured", but then developed symptoms that by 1957, when I first saw her, fitted like a glove with her PBI of 1.5 mcg.%. Her hypothyroidism was treated and within four to six months she began to manifest overt diabetes again.

Blurring of vision is a common complaint of uncontrolled diabetes. This may be due to dehydration of the eyeball or to pathological changes in the cornea, lens or retina. Paresis of accommodation is a frequent finding and lens opacities or cataracts should stimulate the search for diabetes. The typical retinopathy of diabetes consists of hemorrhages, exudates and particularly microaneurysms of the retinal capillaries. Frequently these changes are first noted by the ophthalmologist and he makes the diagnosis without the laboratory tests.

It seems to be agreed that at the same time the retinal capillaries are being affected by the aneurysms, the renal capillaries are undergoing the same degeneration (Kimmelsteil Wilson Dis). Again it must be stressed that these complications can occur prior to the development of overt diabetes or in conjunction with mild glucose intolerance. Collens et al[11] recently described such a case with both retinal and renal complications, who had a normal glucose tolerance test except on one occasion when he developed an infected toe. A stress test with Cortisone proved the existence of diabetes.

The awareness of the occurrence of *neuropathy*, as an initial clinical manifestation of diabetes and recognition of the fact that it can appear before the occurrence of overt diabetes, may help solve some obscure clinical problem. Its clinical appearance may be in the form of a peripheral neuropathy of the extremities, pseudotabes, argyll-robert-

son pupil and other manifestations. Pomeranze[9] recorded 114 cases of neuropathy appearing prior to clinical diabetes. Eighty-five had neuropathy of the lower extremities, 11 had typical diabetic diarrhea and 14 had evidence of sexual failure. Loss of libido and impotence is not an uncommon complaint in diabetes and when noted should stir the index of suspicion. A 45-year-old white male was recently admitted to the hospital with complaints of fatigue, weakness, depression, prostatitis and loss of libido. The urine showed 50-75 WBC per Hpf; blood cholesterol was 290 mgms.% and a fasting blood sugar was 97 mgms.%. The RAI was 35% and a PBI was normal. A glucose tolerance test was done (Fig. 7) and the two

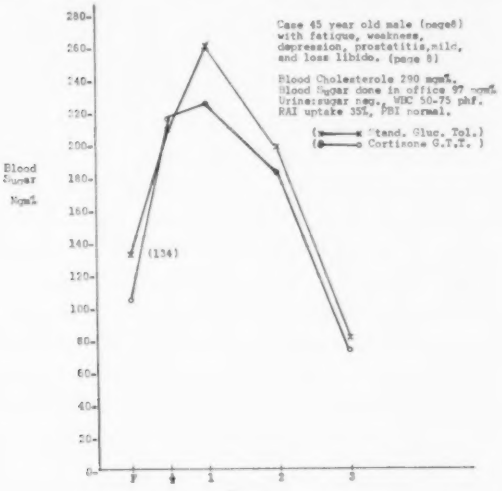

Fig. 7

hour specimen remained elevated, although the three hour blood sugar was normal. The diagnosis of diabetes mellitus was made and with Orinase and weight loss this patient's symptoms abated to a large degree and his libido returned.

Pomeranze[9] states that all cases of sudden paralysis of the muscles supplied by the third, fourth and sixth cranial nerve should be investigated for and if necessary followed for the development of diabetes.

Coronary and peripheral arteriosclerosis with their manifestations of pain and vascular occlusions have been outstanding features of diabetic morbidity and mortality. Their increased frequency in association with the diabetic state, in contrast to the non-diabetic population, again warrants the consideration of glucose intolerance when handling such a patient. A brief account of an example of this is a white male, age 64, who over a period of many years developed repeatedly respiratory infections, complained more and more of weakness, fatigability, anorexia and weight loss and in 1956 developed intermittent claudication. Then he went on to manifest hypertension, coronary arteriosclerotic myocardial disease with abnormal electrocardiographs. He was hospitalized on several occasions during the years between 1956 and 1960 and such ailments as thyroid disease, pheochromocytoma, parathyroid disease, collagen ailments were ruled out. In 1959 a cholesterol was 260 mgms.% and in 1960 reported as 320 mgms.%. Since the PBI and RAI seemed to ruled out hypothyroidism as a cause of the hypercholesterolemia, and even though repeated fasting blood sugars had been normal (87 mgms.%, 114 mgms.% range), a glucose tolerance test was done and proved to be diagnostic of diabetes. (Fig. 8) With Orinase therapy the patient's wife volunteered that he acted more like himself than he had for years.

Another interesting case was that of a 62-year-old white female who was admitted to the hospital with coronary insufficiency with occlusion. She manifested a hypercholesterolemia and a fasting blood sugar of 98 mgms. %. A glucose tolerance test was done and indicated diabetes. (Fig. 8)

The increased incidence of *cholecystitis and cholelithiasis in the diabetic* has long been recognized. This fact alone warrants a thorough screening of all gallbladder patients for diabetics. A study by William R. Jordan, M.D.,[12] led him to the conclusion that gallbladder disease preceded the onset of diabetes twice as often as the reverse was true, and that its duration was an average of 12.9 years prior to the onset of diabetes. This, then emphasizes the importance of follow-up observations in post-operative cases over a long

period of time. The cortisone-stress tests perhaps would indicate a percentage of these diabetics.

A whole new concept concerning *pregnancy and diabetes* has developed within the past few years. As stated by Dr. Shlevin[13] "in the past, many internists and obstetricians have considered the abnormal glucose tolerance test during pregnancy as innocuous. This perhaps has done more harm than

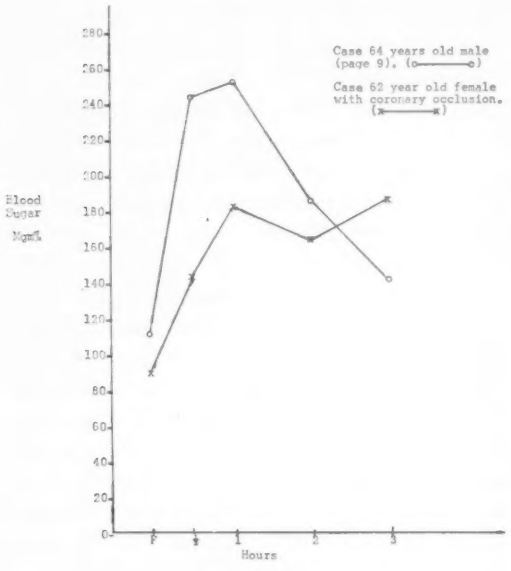

Fig. 8

good by throwing out of focus the proper principles of management of the unrecognized diabetic." The information now gathered plainly points to the fact that fetal and neonatal mortality occurs in extremely high percentage of mothers with pre-diabetes or unrecognized overt diabetes.

A statistical review[14,15] showed the following average findings. History of fetal and neonatal mortality in the range of 20-40% noted in mothers in a period of five years before the development of overt diabetes (in contrast to a control group showing 5%). Large babies of 10 pounds or more have been found to be born to 20% or more of pre-diabetic mothers, often 10 to 20 years before the onset of overt diabetes. Also the pre-diabetic mother has been shown to manifest toxemia in about 10%, abortion 10% and pre-natal loss 30%.

All of this emphasizes the importance of studying the diabetic existence or potentiality in women of childbearing age, particularly if there is a Family History, Poor Obstetrical History or Glycosuria during pregnancy.

Summary

It has been the purpose of this paper to review the subject of the Diagnosis of Diabetes Mellitus. The case reviews were used to impress the fact that they do exist in our practice and not just in the text book. It has been emphasized that the physician cannot depend on the "Text Book Picture" before making the diagnosis. We must utilize our routine urine examinations and more frequently the routine blood sugar, however we must remember their limitations. Much more important is the awareness of the pre- or potential diabetic and the often unrecognized dif-diabetic. This awareness and a good clinical knowledge of the disease create a "High Index of Suspicion" without which many diabetes will go undiagnosed. Then comes the employment of the various diagnostic tests until one can definitely state: There is no Diabetes. (Table #1 summarizes many of the points mentioned in the paper.)

TABLE #1

SUMMARY OF THE SYMPTOMS, CLINICAL FEATURES AND COMPLICATIONS WHICH SHOULD CREATE A HIGH INDEX OF SUSPICION IN THE DIAGNOSIS OF DIABETES

SYMPTOMS

- Weakness-Fatigue
- Weight loss
- Blurred Vision
- Polyphagia
- Polydipsia
- Polyuria
- Dysuria
- Pruritus Vulvae
- Loss Libido

CLINICAL FEATURES

Integument and mucous membrane alterations, infections and dermatoses:

- Dry skin
- Dry mucous membranes
- Furunculosis
- Nail infection (especially fungous)

Delayed healing of lacerations and surgical wounds
Vaginitis
External Otitis Fungous Infection
Xanthalasmia
Necrobiosis Lipoidicum Diabeticorum
Xanthoma Diabeticorum

Systemic chronic resistant infections:
Pulmonary infection
Pyelonephritis
Cystitis
Prostatitis

Miscellaneous:
Family History of Diabetes
Obesity
Hypoglycemic syndrome
Hypercholesterolemia
Post therapeutic intravenous glucose finding of urine sugar.

COMPLICATIONS

Retinopathy—Capillary aneurysms
Cataracts
Nephropathy
Neuropathy
Abnormal Pregnancy
Arteriosclerotic Heart and peripheral vascular disease.
Cholecystitis with lithiasis

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The Case Against Cholesterol in Atherogenesis

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The author doubts that an elevated blood cholesterol is important etiologically in atherosclerosis, feeling rather that it is a result, not a cause, of disease.

HOW SUSCEPTIBLE have we become to the current widespread information concerning cholesterol in atherogenesis? Have we swallowed it hook, line, and sinker, or have we paused to analyze it in the light of pertinent and obviously inconsistent data?

Atherosclerosis consists of two components: the atheroma, a plaque-like intimal deposit of neutral fats, cholesterol, lipophages, and occasionally blood and other evidence of hemorrhage; and the sclerotic phase which represents a physical change of the arteries. It is not clear whether or not the two anatomical components are related. The atheromatous component is thought to be increasing, although the cause for this is not apparent. Above normal elevation in serum cholesterol has been implicated as an important etiological factor. It is felt by the writer that undue emphasis may have been placed upon this substance.

The case for cholesterol in atherogenesis has been presented. Scientific and lay literature abound with information and discoveries which show correlation between the two. One could write a book on these discoveries and not begin to enumerate them all. Too much of this substance is considered, by some, the father of death; too little is not considered pathologic. Yes, this molecule has gained our respect and few doubt its importance.

In medicine we associate the abnormal with pathology; however, in atherosclerosis we have decided that a normal cellular substance, in a slightly high elevation, is capable of producing disease. Disease is usually caused by pathological substances, not normal cells or cellular substances. For example, in leukemia there is a pathologic white cell in the leukocytosis. In contrast, the physiologic leukocytosis in lobar pneumonia is desirable. Could not a pathological lipid be involved in atherogenesis?[1]

Cholesterol has gained its exalted position in atherogenesis by presenting a good case. We could allow witness after witness to bulletin his claim; however, we shall confine our attention to only part of the evidence.

The first witness is the pathologic lesion. Analysis of the atherosclerotic lesion reveals it to be rich in cholesterol. The cholesterol, phospholipid, and neutral fat ratios, however, are the same as those of the blood plasma. Why should cholesterol be signaled out in preference to other lipids? Furthermore, does its presence predicate its guilt? Guilt by association is circumstantial. It does not control judicial thinking; therefore, why allow it to distort medical thinking?

We do not consider hyperglycemia as the etiological factor in diabetes mellitus; hypercalcemia certainly is not the cause of hyperparathyroidism. I have heard no one state that Bence Jones protein causes multiple myeloma. Why the paradox with cholesterol? Hypercholesterolemia is the result of disease, not necessarily the cause.

The next witness testifies that atherosclerosis can be simulated in several animal species by experimental conditions which disturb lipid metabolism; (i.e., by administration of cholesterol or bile salts, derangement of thyroid function, etc.) Anitschow first

published his classical experiment in 1913 and this type of experimental approach has since absorbed the attention of scientists; notwithstanding, many now doubt its importance. Since neither man nor beast ingests cholesterol in the pure state, the administration of cholesterol by Anitschow amounted to poisoning.[1] The rabbit lesion consists of lipid deposition in the intima of the intramyocardial part of the coronary artery. This location is usual in human disease. Furthermore, the animal lesion was devoid of the sclerotic component. The atherogenic-inducing potentialities of the same diet differ in different species. The fact that a certain diet has been found atherogenic in one species is not prima-facie evidence that this will be obtained in other species. Dr. Samuel Gordon of Lederle Laboratories has reported that the chipmunk-sized gerbil has remained atheroma-free on a one per cent daily cholesterol diet—far in excess of what an average man ingests daily. The serum cholesterol in sixty gerbils which were studied reached a peak of 1,200 mg. per 100 c.c. about a week after the one per cent diet began. Later, blood cholesterol leveled off at about 600 mg. per 100 c.c. After six months thoracic and abdominal aortas of gerbils were examined microscopically. In no case was there any evidence of atheroma formation. No correlation, pertaining to humans, should be attached to Anitschow's experiment.

The next witness is a group of diseases in which there is a high serum cholesterol level and also a higher rate of atherosclerosis. Diabetes mellitus, nephrosis, myxedema, xanthematosis, and familial hypercholesterolemia fall into this category. Conversely, in thyrotoxicosis, a disease associated with hypolipemia, there is a decreased incidence of atherosclerosis. In idiopathic steatorrhea, with impaired dietary absorption of fats, atherosclerosis is seldom seen. Innumerable studies correlate hypercholesterolemia with angina pectoris. Data to substantiate this correlation is endless. This witness has presented strong evidence—but is the only similar abnormality among these conditions hypercholesterolemia? Isn't it plausible to assume that there are other abnormalities, that are common to all, with which we are not familiar? We know that if there is one physical anomaly there is greater risk of multiple anomalies. Similarly, I feel that it is reasonable to assume that a child born with a potential carbohydrate metabolic deficiency would also be prone to develop a metabolic lipid or protein deficiency. The increased relationship between diabetes, gout, and hyperlipemia would fortify this theory. Some diabetics escape the lip anomaly and, clinically, this is vertified. Hypercholesterolemia should be expected to co-exist in many diseases!

Diet, as an etiological factor in atherogenesis, has been given considerable impetus by Ancel Keys and his associates.[2] Worldwide studies have demonstrated a relationship between the level of serum cholesterol and dietary fat intake. Those populations with a higher percentage of fat in diet have a higher incidence of atherosclerosis. It is likely that, in a susceptible individual, diet could speed the atherosclerotic process, or any process; however, there are those who do not develop premature atherosclerosis regardless of diet. Witness the obese, senescent glutton who is continually signaled out by those patients who do not wish to diet. If diet is an important etiological factor in the development of atherosclerosis we must pity the poor ulcer patient who must exist on his atherogenic diet! To draw etiological conclusions from worldwide dietary studies is misleading since there are genetic predisposition and protection. Of children with one hypercholesterolemic parent, 17% are hypocholesterolemic; when both parents are normal cholesterolemic, only 2% have high serum levels.[3] Many feel that hyperlipemia is an endocrine disorder and that diet would be no more important in its development than diet is an etiological factor in diabetes.[4] Keys has tried to correlate the lack of atherosclerosis among the Bantu with their low fat diet. The American Negro, who has a generous dietary fat intake, has a markedly

less incidence of coronary heart disease than his white counterpart. The Negro apparently is sickle cell and tubercular susceptible; but in retribution, God made him atherosclerotic resistant. I do not wish to discredit the potential value of Key's work but there are variable factors in diet which he fails to consider. The possibility of an anti-atherogenic factor in diet (like the intrinsic factor in pernicious anemia) is not mentioned. The intima of the Bantu's arteries has not been exposed to the many and varied chemical compounds, such as penicillin, sulfa, food preservatives, vitamin-enriched foods, tetracyclines, etc., as has the intima of his white counterpart. Could not the increase in atherosclerosis be due to the increased consumption of one of these substances? As remote as it may seem, this hypothesis can be argued statistically. Either this observation is true or it reveals the fundamental weakness of a purely statistical approach to pathologic events.

The influence of sex hormones on atherogenesis has been cited as evidence. Women, particularly young women, possess some degree of protection from atherosclerosis and its more serious complications. Oophorectomized women lose this protection. Administration of estrogen to men who have survived one or more attacks of myocardial infarctions accomplishes several actions on the abnormal blood lipids found in these men.[5] Since many hormonal substances are involved in lipid metabolism (pancreas, adrenals, and thyroid) it is unlikely that lipids are influenced, primarily, by sex hormones. Wolffe feels that atherogenesis could be a metabolic disease; it is his opinion that the vascular complications of diabetes mellitus can be attributed to the present manufacture of "pure" insulin. In the present manufacture many protective hormonal factors of the pancreas are being destroyed. The great French cardiologist, Prof. Vaquez, stated that he had never encountered the vascular complication of diabetes mellitus while using crude insulin. These observations, also, have been substantiated by his own studies.[6]

Statistics in many and varied forms have been presented as evidence. They show correlation between super cholesterolemia and atherosclerosis. There is no doubt that physicians are more coronary-conscious and there may be truth in the statement that the death rate from coronary artery disease is directly proportional to the number of physicians per thousand population. The importance of correct diagnosis in relation to statistics becomes doubly apparent when we realize that the margin of error in detection of atherosclerosis is considerable. How does one measure total atherosclerosis? Unfortunately, a little in the wrong location can be fatal and the individual in question becomes a statistic. This is in contrast with the bald hirsute. As baldness is not fatal he receives scant attention. There are no hair-raising funds for his glossy pate! This individual doesn't lack hair-growing ability—he just cannot grow it in the right location! Similarly, the coronary atherosclerotic just doesn't grow his atherosclerosis in the proper location.

Mechanical factors and hemodynamics doubtless contribute to the development of atherosclerosis—a fact which has long been recognized. The pressure maintained in the cardiovascular system must have an important influence in the production of organic changes in its walls. The selective predilection for development of the atherosclerotic lesion fortifies the etiological significance of hemodynamics. Here, also, lies a fertile field for future investigation.

Though scarcely enough attention has been given the intimal cells in atherogenesis, they are undoubtedly important. Could not the pathology be in these cells rather than in that with which they come into contact? I feel that the atheroma may be analogous to the colloid goitre. In colloid goitre the first reaction to iodine deficiency is hypermia, decrease in the amount of colloid and hyperplasia of the thyroid epithelium. Later, there are involuntary changes which include colloid accumulation, fibrosis, cyst formation, hemorrhage, and calcification. One

readily sees some gross similarity between the two pathologic conditions.[7]

Sometimes we must look beyond the apparent because the apparent may not be real. We must be aware of the obscure and seemingly unrelated cause-and-effect. The medical treatment of atherosclerosis has already been littered with carcasses of premature and false claims. I am sure that when the final answer is discovered we shall find that many treatments have been harmful. We now realize the danger from the unfortunately promiscuous use of adrenal steroids and antibiotics—a situation which was not foreseen.

It is difficult to change when one has accepted the basic premise of a system. We are taught that there is an answer to everything, but by accepting the imperfect reality we may often do better. In "Novum Organum", Sir Francis Bacon established first his doubt of all things before proceeding to a consideration of knowledge. "In general," he said, "let every student take this as a rule —that whatever his mind seizes and dwells upon with peculiar satisfaction, is to be held with suspicion; and that so much care is to be taken; in dealing with such questions, to keep understanding even and clear. Doubt all things before you believe anything? Watch your idols." Could this be true with cholesterol? I feel that we must proceed with caution when we manipulate the cholesterol molecule. We should not be too readily swayed by those who demand that the public be told "the truth" while we are still attempting to find the answer. We should be wary of explaining the increase of atherogenesis when the etiology is not known. I do not disagree with those who advise lowering elevated serum cholesterol—but let us limit our methods. The widespread publicity given to diet will burden many with fad diets.

The constant search for and introduction of new products by the pharmaceutical industry is ample evidence of the lack of uniformity of opinion and satisfaction with this subject. New products have been, and will continue to be, introduced at a rapid rate. Let us keep our reason clear and not shift from product to product with each touted claim.

Far too few realize that behind the headlines a scientific debate is raging!

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Keratoplasty

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Corneal grafting is not only useful in restoring vision but also in the treatment of certain eye diseases. It is to be hoped that donor eyes will become increasingly available to expedite this work.

UP UNTIL RECENT YEARS keratoplasty could only be done satisfactorily in the larger medical centers. During the last few years, more Eye Banks have been established throughout the country* and donor eyes are now more freely available to the ophthalmologists in smaller centers. This improved availability has been of benefit to the patient unwilling to travel distances to have the necessary surgery and also to those patients that have had keratoplasties done to arrest rapidly worsening corneal diseases.

Keratoplasty can be divided into two basic techniques: first, the penetrating and second, the lamellar graft. In penetrating grafts, the whole thickness of the cornea is replaced. Most operators restrict the size (diameter) of the graft to within certain limits (5-8 mm).

In our series, the most satisfactory results were obtained in the 6-7 mm. grafts. Originally, keratoplasty was done almost entirely for optical purposes, i.e., to improve vision. As the field and techniques have advanced, more uses for keratoplasty have been found and the field of therapeutic keratoplasty has been enlarged. The conditions that respond most satisfactorily are keratoconus (90-95%), nodular and lattice dystrophy (80%), interstitial keratitis (60%), disciform keratitis (60%), leukomas and pyogenic ulcers (50%).

In the following series, several cases were done for visual improvement primarily; several others for therapeutic reasons. We have twelve patients (13 eyes) that were done for visual improvement and eight that were done for therapeutic reasons. Those done for visual improvement included keratoconus, nodular dystrophy (Groenouw's), dense leukomas following injury or corneal infections. If the patient's vision fell below 20/200, in bilateral disease, keratoplasty was indicated. In those cases with unilateral scarring, a preoperative visual acuity of 20/100 or less, surgery was indicated. In most of these cases, a penetrating keratoplasty was performed. The following techniques was used:

1. Mild sedation, e.g., 75 Demerol, Nembutal gr. 3/4, Phenergan 25 mgm.—one hour before surgery.
2. Diamox 250 mgm.—three hours before surgery and repeated one-half hour before surgery.
3. Irrigation of lacrimal sacs with normal saline.
4. Instillation of Pilocarpine, 2%, four to six times before surgery to produce miosis. Starting two hours before surgery.
5. Sterile pre-operative preparation.
6. Akinesia, Van Lint and retrobulbar, using Xylocaine 1%.
7. Lid sutures were used to retract lid. 40 black silk fixation sutures inserted through superior rectus and inferior rectus.

*Eye Bank and Sight Conservation Society of Virginia, 711 South Jefferson Street, Roanoke.

The graft was removed from donor eye with a Castroviejo trephine and similar button was removed from recipient eye. Graft was positioned and sutured in place with Virgin Silk (10-0) direct appositional sutures, (15-24 sutures), depending on the size of the graft. Air was inserted into anterior chamber with 27 gauge needle. The pupil was either constricted or dilated to hold the iris from the incision. One eye was bandaged only. Bed rest the first four days, then patient was ambulatory. Sutures were removed on the twenty-first day. Patients were kept on antibiotics and steroids as required.

Summary of Cases Done Primarily For Visual Improvement

Case #1, Mr. G. A., age 52, had had repeated attacks of herpetic keratitis in O. S. over the last four years with severe scarring of the cornea, and with severe visual loss. Vision had been reduced to light perception and large objects only. He had had no further ulcerations or flare-ups of the herpetic condition for one year before time of surgery. Mr. G. A. underwent a penetrating keratoplasty using a 6 mm. graft with direct appositional sutures. His recovery was uneventful and at the time of discharge, his vision was 20/70 in the affected eye. After eighteen months, his vision had improved to 20/60 and the graft is completely clear.

Case #2, Mr. I. M., age 47, had a long history of visual difficulty dating back from his early twenties. When examined, he had bilateral keratoconus and pre-senile cataracts in both eyes. His vision, at the time of surgery, was reduced to 20/400 in O. D. and light perception and large objects only in O. S. It was decided to do the patient's left eye first, using a penetrating keratoplasty and in six months to do a cataract extraction. A 7 mm. penetrating graft was done on his left eye, using direct appositional sutures. He had an uneventful recovery, the graft remained clear and in six months the graft was completely clear when he was re-admitted to have a cataract extraction. He had an intracapsular extraction with a wide iridectomy, and once again he had an uneventful recovery from this. Fortunately, the graft remained clear and at the present date, two months after his operation, his vision is 20/20 in his left eye with correction. He is returning for similar procedures on his right eye.

Fig. I. Case # 2, I. M. Post-operative. Patient had pre-senile cataracts and keratoconus, O. U.
O. S. 7 mm. penetrating graft and intracapsular extraction and iridectomy.

Case #3, Mrs. H. H., age 48, also had keratoconus in both eyes and history of visual difficulties over the last fifteen years. When admitted for surgery, she had vision of count fingers at ½ meter in either eye which could not be improved either by lenses or contact lenses. It was decided to do a penetrating graft on her right eye and a 7 mm. penetrating graft, with direct appositional sutures, was performed. She had an

Fig. II. Case #3, Mrs. E. H. Pre-operative. Showing advanced keratoconus, O. D.

uneventful recovery and was discharged from the hospital at the end of one month. Her vision at the end of one year was 20/50, without correction, in her right eye. It could be improved to 20/40 with correction and a contact lens at a later date will probably improve this further. She was re-admitted two months before this article was written and a 7 mm. graft was done in her

Fig. III. Case #3, E. H. Post-operative. Showing 7 mm. penetrating graft.

left eye. The graft is progressing satisfactorily, is gradually clearing and we hope that she will have satisfactory vision in her left eye as well.

Case #4, a man, age 56, had keratoconus, much more marked in his left eye. He had not had satisfactory vision in this eye for ten years. On examination, V. O. D. was 20/40, V. O. S. was count fingers at ½ meter. A 7 mm. penetrating graft was done, with appositional sutures, on the left eye. He had an uneventful recovery. The graft was cloudy at first but has gradually cleared and when this patient was discharged, his vision was 20/200. He was discharged from our care to be followed by his local ophthalmologist. We do not have a final visual acuity on this patient.

Case #5, Mr. O. J., age 48, had bilateral Groenouw's distrophy of the granular type. V. O. D. was count fingers and V. O. S. was count fingers. He had a 6 mm. penetrating keratoplasty, O. S. Four weeks postoperatively he developed secondary glaucoma, which was controlled by iridectomy. At first the graft remained clear, but over the next three months it gradually became cloudy. His vision was not improved. This represents a failure and the patient should have a re-graft.

Case #6, Mr. B. F., age 46, who also had Groenouw's dystrophy. Vision was count fingers at one meter in either eye and he had a 6 mm. penetrating keratoplasty, O. S., with direct appositional sutures. His vision at the end of three months was 20/70 and at the end of two years the graft is still clear.

Case #7, Mr. H. W., age 59, had a severe injury to his left eye which had resulted in a corneal ulcer and dense leukomatous formation which had been present for approximately twelve years. His vision at the time of operation was light perception only. He had a 6 mm. penetrating keratoplasty with

Fig. IV. Case #7, H. W. Post-operative. 6 mm. penetrating graft for central leukoma, O. S.

direct appositional sutures. His vision, at the end of three months, was 20/60, with correction, and gradually improved over the next four months to the neighborhood of 20/40. At the end of 2½ years, the graft is still clear. It is interesting to note that this patient, eight months after he had the keratoplasty, developed an optic neuritis in his right or good eye, with complete loss of vision, decreasing his vision to 20/400 so the patient had to rely on his operated eye. The neuritis cleared; however, his vision has remained in this eye at about the 20/40 level. I think this is one of the best arguments that we have seen for operating on patients that have one good eye and one that has a corneal

lesion that could be benefited by keratoplasty. This patient, if he had not had the keratoplasty, would have been completely incapacitated for the time that he had the optic neuritis.

Case #8, Mr. M., age 41, also had a severe injury to his left eye in childhood resulting in a dense central leukoma involving the full thickness of the cornea. Vision at the time of operation was V. O. D. 20/30 and V. O. S. light perception only. He underwent a 7 mm. penetrating keratoplasty and at the end of three months his vision was 20/200. He was discharged to his local ophthalmologist and we do not have a final visual acuity on him.

Case #9, Mr. F., had had a severe injury to both eyes from chemical keratitis with opacification of the whole cornea. The right eye had been removed ten years before. In the left eye the whole cornea was severely scarred, involving the full thickness of the cornea. He had a complete opacification of the cornea and had received radiation three

THOSE DONE FOR VISUAL IMPROVEMENT

Patient	Age	Indications	Type of Graft	Pre-Op Visn.	Post-Op Vision With Correction	Overall Result
H. W.	59	Central leukoma, O. S.	6 mm. Penetrating Keratoplasty	Light perception	20/60 2½ years	Improved
A. M.	41	Central leukoma, O. S.	7 mm. Penetrating Keratoplasty	Light perception	20/200	Improved
W. F.	50	Chemical keratitis with complete opacification of cornea, radiation cataract	8 mm. Penetrating Keratoplasty Cataract extraction 6 months later	Poor light perception	Poor light perception	Unimproved
G. S. A.	52	Central leukoma from repeated herpetic keratitis	7 mm. Penetrating graft	L. P's.	20/80	Improved
I. M.	47	Keratoconus Pre-senile cataracts, O. U.	7 mm. Penetrating graft, O. S. 6 mos. later intracapsular cataract extraction	Light perception and hand movements	20/20	Improved
H. H.	48	Kerataconus, O. U.	7 mm. graft, O. D. Penetrating	Count fingers ½ M.	20/60	Improved
H. H.	48	Kerataconus, O. U.	7 mm. penetrating graft, O. S.	40/400	20/100 6 wks. post-op.	Improved
I. W.	56	Kerataconus, O. U.	O. S., 7 mm. penetrating graft	O. D.—20/40 O. S.—Count fingers ½ M.	V. O. S. 20/200 6 wks. Post-operative No follow up	Improved
O. J.	48	Groenouw's Dystrophy, O. U.	6 mm. penetrating, O. S.	L. P's. and hand movements	Graft cloudy L. P's.	Worsened
B. F.	46	Groenouw's Dystrophy, O. U.	7 mm. penetrating, O. S.	L. P's. and hand movements	20/70—4 mos. Latest follow up	Improved
T. D.	65	Bilateral iridenclesis Cataract extraction, O. D. O. S.—Dense corneal leukoma, senile cataract	7 mm. lamellar keratoplasty	Hand movements and L. P.	Count fingers	Improved
R. S.	44	Dense central leukoma, O. S.	7 mm. penetrating keratoplasty, O. S.	Light perception	20/70 4 wks. post-op.	Improved

years before to decrease the vascularization of the cornea. This had, in turn, produced what appeared to be a radiation cataract; however, this cataract was not visible until after the time of surgery. He had an 8 mm. penetrating keratoplasty with freeing of anterior synechia. This graft remained clear and at the time of surgery, the dense cataract was visualized. His preoperative vision was light perception only, the graft remained clear and at the end of six months the cataract was removed. Following the cataract extraction, the graft became cloudy and even after a re-graft, it remained cloudy. His vision at the time is still light perception.

Case #10, Mrs. D., age 65, had chronic open angle glaucoma for fifteen years and had had bilateral iridenclesis and a cataract extraction, O. D. This had resulted in a Bullous Keratitis, O. D. Light perception was present in this eye. In her left eye she had a dense corneal leukoma, probably in the nature of a corneal dystrophy from a long-standing glaucoma. She also had a moderately advanced cataract. Vision was hand movements and count fingers, poorly, at ½ meter in this eye. The leukoma involved the outer ⅔ of her cornea so a lamellar keratoplasty, 7 mm. in diameter, was done and this graft has remained clear and her cornea has cleared considerably. At the present time we are waiting for approximately eight months to a year before we do a cataract extraction. With her long-standing history of glaucoma, and her difficulties with her right eye, the chances of her getting a good result are slim but still, with her vision the way it is, there is very little to be lost.

Case #11, Mr. S., age 42, had an injury to his left eye fifteen years before. Three years following this injury he had severe ulceration of his left eye which resulted in a dense leukoma involving the full thickness of the cornea. His vision, when seen here, was light perception only in O. S. and 20/25, O. D. He had a 7 mm. penetrating keratoplasty with direct appositional sutures. This completely replaced the corneal scarring and at the end of six weeks his vision has improved to 20/80.

In the patients that were operated on for improvement of their vision, earlier cases were done using the outlined technique but using 6-0 black silk with Grieshaber needles. These sutures, of course, are fine but we found they produced considerable corneal irritation and it was not until two years ago, when we switched over to 10-0 Virgin Silk, that we noticed a marked improvement in our operative results. There was much less irritation present in the eye, sutures could be left in longer which resulted in much better healing and apposition of the keratoplasty wounds, and when they were removed there was much less chance of collapse of the anterior chamber. Our visual results follow pretty well the pattern established elsewhere. The results in keratoconus were excellent, the results in corneal dystrophy were about 50%, and the results in corneal leukomas in the neighborhood of 70%. In the earlier cases we had to rely on eyes shipped in from New York and elsewhere and very often these eyes were used at almost the end of the 48-hour period. In the later cases that we have done, we have obtained eyes locally and used them within 12 hours. We found that the grafts have remained much clearer earlier, and the overall results are more satisfactory. The finding, of course, applies only to the penetrating types. We have not noticed any difference using fresh eyes or older eyes in the lamellar keratoplasties.

Therapeutic Keratoplasty

Therapeutic keratoplasty is developing into a most useful and gratifying procedure in treating chronic and recurrent diseases of the cornea. Herpetic infections of the cornea have increased in incidence and importance. It is now the most frequent cause of visual loss due to corneal opacities and is becoming the most frequent indication for therapeutic keratoplasty in this country.

Herpes simplex of the cornea responds

poorly to most treatment. In our practice, the number of severe cases with deep stromal involvement has increased over the last two years. In the recurrent cases, the only satisfactory method is the total removal of the involved cornea by keratoplasty.

In Herpes, the transplants were done for several reasons. Usually, the transplants are indicated in recurrent episodes of superficial keratitis either diffuse or circumscribed, or deeper lesions recurring over many months. In active cases with deep stromal involvement with threatened perforation, grafting is definitely indicated. The removal of the diseased tissues has a beneficial effect through several suggested ways. It removes the infecting virus and the necrotic tissue associated with it. The graft possibly introduces fixed antibodies to the host cornea also. Felatov believes the introduction of new tissue has a beneficial biological effect. Vascularity is also reduced which brings about a diminution of the allergic reaction. Therapeutic grafting has been beneficial in certain types of perforating ulcers (bacterial and fungal) and at times this technique may be an outright emergency. The technique followed in our cases is basically the same as the perforating keratoplasties except only a partial thickness of the cornea is removed (0.5 to 0.6mm). The postoperative recovery period is much shorter and most of these people can return to work within 2-3 weeks.

Corticalsteroids are contraindicated in herpes simplex; however, steroids are a great therapeutic tool in corneal grafting. They have been used in three of the cases done in the series with beneficial effect and as yet no untoward effects.

Probably some of the most satisfying surgery that we have done has been in the field of therapeutic keratoplasties:

Case #1: Mr. H., age 50, who had had a bilateral cataract extraction which had resulted in a Bullous Keratitis with almost intractable pain in his left eye. He had the choice of having his left eye removed or trying a therapeutic keratoplasty. His vision in this eye before surgery was light perception and hand movements. We explained to the patient that chances of his getting a satisfactory result through keratoplasty were not good, but with the other alternative, he was anxious to try the corneal graft. A 7 mm. penetrating keratoplasty, with direct appositional sutures, was done. The patient had an uneventful recovery, the sutures were removed on the twenty-first day. The graft remained semi-transparent; however, his vision was improved to count fingers at ½ meter. The most remarkable thing in this case is that he had complete relief of pain and this has continued over the last year. How long it will continue, we do not know; however, the patient is well satisfied and we have not had to remove his eye as yet.

Case #2, Mr. E., age 64, who was admitted here with a perforated corneal ulcer, had ulceration of his cornea and had been on ophthalmic solutions which combined antibiotics and steroids for three weeks before being seen. When admitted, the ulcer had already perforated and we thought probably that the ulcer had been due to a mycotic infection. No bacteria could be cultured from it and no fungi were cultured. We debated whether the eye should be removed because an early endophthalmitis was present. We had at this time an extra eye, left over from another corneal transplant, which was three days old and had exceeded the suggested time limit. A 7 mm. penetrating graft was done which completely removed the corneal ulcer and during the operation the prolapsed and adherent iris was excised. The graft, to our surprise, took very satisfactorily; it did not remain perfectly clear, it was semi-transparent; however, the general condition of his eye improved and the infection subsided. He does not have any useful vision in his eye at present and after eight months it is still injected and probably the safest course to follow would be to enucleate the eye.

Case #3, Mr. T. D., age 40, had a chemical burn to his left eye which resulted in a symblepharon, approximately the 5 o'clock position. This had recurred despite

several removals. The inferior cul-de-sac had been restored by a conjunctival graft but he still had an adhesion on to the cornea. He was admitted and an elliptical piece of cornea containing the symblepharon was removed. A similar elliptical piece of donor cornea was grafted in its place. The thickness of the cornea that was grafted was approximately ⅔ the corneal thickness. It was held in place by direct appositional sutures to the cornea and to the sclera. His vision before surgery was 20/30 and after two months his vision was 20/25. The important thing here is that the symblepharon has not recurred and the lamellar graft has remained perfectly clear. We feel that this is an ideal procedure for this type of injury and also in recurrent pterygiums. It is relatively free of complications and does not carry the danger of a penetrating graft.

Case #4, Mr. W., age 30, who had had a corneal dystrophy, O. D., since birth and apparently had had a congenital rupture of Descemets membrane which had resulted in a chronic Bullous keratitis. He had gotten along fairly well up until one year before the operation, at which time he started having repeated erosions of the corneal epithelium and ulceration. This resulted in severe pain and over the last three months the patient had been unable to work. He had light perception and hand movements only in this eye. Vision in his left eye was 20/20. He was advised that he might benefit from a keratoplasty; however, the chances of its improving his vision were not good, but it might result in relief of pain. He had an 8 mm. penetrating graft and he made an uneventful recovery. To our surprise, the graft gradually cleared and at the present time his vision is 20/400 in this right eye. He had a dramatic relief of pain and at the end of six months has had no ulceration.

Case #5, Mr. T. S. D., age 65, had had repeated violent infections of the corneas of the right eye. These were herpetic and resulted eventually in a disciform keratitis of a severe nature which resulted in complete opacification of the superficial layers of the cornea, actually the outer half of the cornea. He kept having repeated ulcerations and pain. Because of this, we advised him to have

THERAPEUTIC GRAFTS

Patient	Age	Indications	Type of Graft	Pre-Op Visn.	Post-Op Vision With Correction	Overall Result
T. D.	40	Symblepharon recurrent	6 mm. lamellar keratoplasty, eccentrically placed	20/30	20/25 No recurrence	Improved
S. W.	30	Congenital corneal dystrophy, rupture of descemets membrane Recurrent ulceration	8 mm. penetrating graft, O. D. O.D.	L. P's.	20/400	Improved
T. S.	65	Disciform keratitis severe, recurrent ulcerations	8 mm. lamellar graft	L. P's.	Count fingers No recurrence (5 mos.)	Improved
M. C.	39	Disciform keratitis (18 mos. duration)	7 mm. lamellar graft, O. D.	L. P's. and hand movements	20/70 (6 wks.) No recurrence	Improved
C. G.	30	Repeated attacks, herpetic keratitis O. D. for 5 years	8 mm. lamellar graft, O. D.	Count fingers O. D.	20/70 (7 wks.)	Improved
H. H.	50	Bullous keratitis O. S. Post cataract Intractable pain	7 mm. penetrating keratoplasty	L. P's. and hand movements	Count fingers ½ M. Relief of pain one year	Improved
C. E.	64	Perforated corneal ulcer, O. S.	7 mm. penetrating, O. S.	L. P's.	L. P's.	Temporary improvement

a keratoplasty. An 8 mm. lamellar keratoplasty was done, held in place with direct appositional sutures. His vision before surgery was light perception only and he made an uneventful recovery, his cornea has cleared remarkably, at the present time he counts fingers and at the end of four months has had no recurrence of his ulceration and no recurrence of his pain. Over the next few months the graft will probably clear even more and, if his eye stays quiet, we plan to do a penetrating keratoplasty to improve his vision.

Case #6, Mrs. M. C., age 69, had a severe disciform keratitis, O. D., for 1½ years and in spite of various treatments here and elsewhere, no relief could be obtained. She had intractable pain which had made it impossible for her to work or carry out her duties. She was admitted to the hospital and a 7 mm. lamellar keratoplasty was done, completely removing the disciform ulceration. Her vision before surgery was light perception and hand movements, and at the end of two months her vision is 20/70. The cornea has healed nicely and she is completely free of pain.

Fig. V. Case #6, Recurrent herpetic keratitis, O. D. (5 years)

Case #7, Mr. G., age 30, had repeated attacks of herpetic keratitis involving his right eye over five years time. Over the last year he had had six attacks of herpetic keratitis which necessitated his stopping work, and even with conventional treatment he still had pain which at times required narcotics. He was advised to have a keratoplasty and was admitted to the hospital. An 8 mm. lamellar keratoplasty was done. His vision, before surgery, was count fingers in his right eye and 20/20 in his left. At the end of six weeks, his vision is 20/70 and improving. He has had a dramatic relief of pain and is back to his old occupation. His cornea is completely free of ulceration and in the patient's own words: "It is the first time in five years that my eye has felt like a normal eye."

The common indication in our series was herpetic keratitis. We found this a most useful tool in the treatment of these diseases and the only satisfactory one we have at the present time. Improvement of vision was not remarkable in these cases, but the relief of pain was, and these patients are probably the most satisfied of any of the keratoplasty patients we have had. Some of these cases in the therapeutic series were done because the only alternative was enucleation and we feel that we have rescued several eyes which otherwise would have been removed.

The complications in keratoplasty are frequent, especially in the penetrating type of graft. Clouding of the graft is a constant

Fig. VI. Case #6. 8 mm. lamellar graft (therapeutic) replacing the involved tissue.

worry and seems unrelated to surgical technique, although if the graft edges are sharp and in good apposition, the incidence of clouding is decreased.

In our early cases we had some difficulty with the edges bulging in parts of the graft. We were removing the sutures on the fourteenth day and were using 60 black silk. Since using virgin silk and leaving the sutures

in for twenty-one days, we have not been plagued by this difficulty.

In one case, secondary glaucoma developed which was controlled by iridectomy but the graft has remained cloudy.

In the treatment of herpetic keratitis the complications were nil and the results in these cases are second only to the results in keratoconus.

In the one case of perforated ulcer, the patient has maintained his eye but it has remained irritated and probably will be enucleated in the future.

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Less TB and Increasing Lung Cancer

A decline in deaths due to tuberculosis has been suggested as a factor in the increasing occurrence of lung cancer.

Writing in the December 31st Journal of the American Medical Association, Drs. Broda O. Barnes, Denver, and Max Ratzenhofer, Graz, Austria, said more persons were surviving TB early in life only to contract lung cancer in their later years. They predicted that "a large increase" in lung cancer could be expected as tuberculosis deaths continue to decline.

Their conclusions were based on data from 26,546 autopsies performed during the past 15 years at the University of Graz.

The study showed that "a great reduction in tuberculosis is occurring in Graz, as elsewhere." It also showed that the average age of patients who died from tuberculosis was rising.

"In 1944, the average age at death was 38 years, while in 1957 it was 54 years. In other words, the tubercular patient was approaching the 'cancer age' before death."

A further indication was that TB and lung cancer are not found frequently in the same person at the same time.

It appears "that there are two diseases competing for the same person: tuberculosis at an early age and bronchogenic carcinoma as he grows older."

"It should be expected that countries with a high incidence of tuberculosis would have a low incidence of bronchogenic carcinoma and vice versa, unless other factors are present."

"Our data indicate that a large increase in bronchogenic cancer can be expected as the death rate from tuberculosis decreases.

"At the turn of the century, the United States had about 200 deaths per 100,000 from tuberculosis. If the same proportion of the population is susceptible to tuberculosis today, and if 20 per cent of this same population is susceptible to bronchogenic carcinoma, then 40 per 100,000 might succumb to lung cancer when tuberculosis is eradicated. This, of course, is conjecture, but it illustrates that a further increase in bronchogenic carcinoma may be expected."

Kartagener's Syndrome

R. N. DE NIORD, M.D.
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A case of this unusual syndrome is reported. The patient was treated surgically with considerable improvement.

KARTAGENER'S SYNDROME refers to a congenital bronchiectasis with situs inversus and chronic sinusitis. It is interesting to note that about fifteen to twenty per cent of the congenital dextrocordias are associated with bronchiectasis, which is an incidence infinitely greater than usually present in our general population.[1]

This combination of dextrocardia, bronchiectasis, and sinusitis was first described by Kartagener in 1933. Since that time approximately fifty cases have been so described and reviewed.[2,3]

There is a clear association between mucoviscoidosis of congenital cystic pancreatitis origin and infant bronchiectasis. However, the Kartagener's Syndrome does not seem to be associated with mucoviscoidosis. There is rather a congenital and extremely early appearing basilar pulmonary infection associated with saccular bronchiectasis and severe recurrent pulmonary infections. This triad is so unusual in actual occurrence that this author thought it noteworthy to report an interesting case with description of surgical lobectomy and dramatic improvement.

Case History

D. M.—A ten year old white male who had been chronically ill since infancy, with poor development and recurrent severe pulmonary infections.

This patient had an older sister who also suffered from situs inversus, chronic sinusitis and bronchiectasis. The sister was operated upon approximately four years earlier, but did not survive the surgery.

The bronchograms revealed basilar bronchiectasis on the right side, confirmed by the auscultatory findings of poor breath sounds, wheezes, and rhonchi at the right base. The findings of complete situs inversus with the air bubble on the right side, the liver on the left side, and the heart in the right chest indicated a reversal of the pulmonary anatomy.

Because of the discrete localization of bronchiectasis in the right lower lobe (anatomical left lower lobe) a lower lobectomy was done through the right chest and a completely destroyed atelectatic and bronchiectatic left lower lobe was removed from the right chest. Following surgery the patient did well with an uneventful recovery and

Fig. I. Bronchogram revealing extremely poor filling on the right with beading of the bronchi.

is now one year post-operative. There has been adequate wound healing and a disappearance of the chronic pulmonary infections. The child has now returned to school, is gaining weight, and carries on usual childhood activities for his age.

Discussion

The occurrence of the pan sinusitis in Kartagener's Syndrome makes it impossible to say whether the sinusitis or the bronchiectasis comes first. Both may be a manifestation of an increased susceptibility to infections for reasons unknown to us at the present time. It is known that reduced susceptibility to a disease does occur in congenital malformations as in the Fanconi Syndrome, and certainly it is also recognized that bronchiectasis has been observed in other debilitating diseases, such as congenital heart disease, Kilppel-Feil Deformity, and Polydactyly.[5] It is probable that the usual causes of bronchiectasis, that is, chronic infection,

Fig. II. Postoperative x-ray with catheter in position. Note the heart in the right chest. A left lower lobectomy has been performed through the right chest.

with gross destruction of bronchial walls, and obstruction with a mechanical narrowing of the bronchial lumen produce the end result of bronchiectasis. Why these children with situs inversus are more susceptible is entirely unknown, and probably falls somewhere in the realm of poor "ground substance" or weak protoplasm. These general terms do not adequately or scientifically express a clear understanding of the deficiency, but to the knowledge of this author there is no further adequate explanation. Probably the answer to this problem lies in the determination of serum protein abnormalities and correct evaluation of amino acid and fibrinogen synthesis in the determination of ground substance quality.

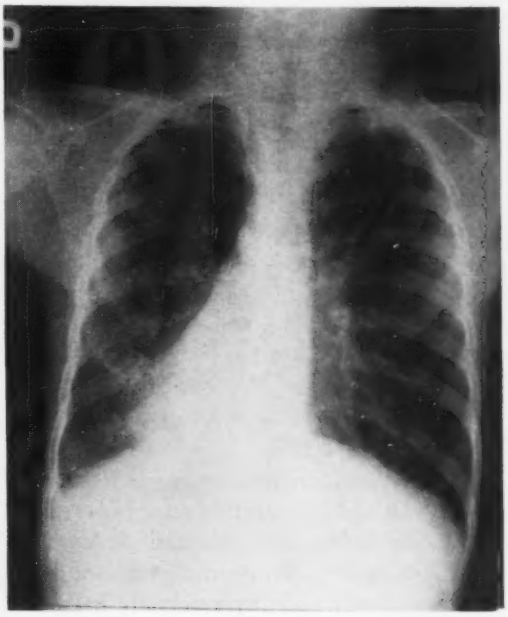

Fig. III. Chest x-ray one year postoperatively revealing essentially clear lung fields. Note that the heart is retracted further into the right chest.

Summary

1. A description of Kartagener's Syndrome is given with a case illustration in which a successful resection of the bronchiectatic lobe was performed.
2. A brief discussion of the syndrome is presented with a review of available information.
3. It is felt that significant relief from this congenital abnormality can be obtained if there is adequate localization of the bronchiectasis affording an op-

portunity for resection and removal of the diseased portion of the lung.

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Fights Congestive Heart Failure

The mechanical kidney has become a weapon against heart disease. An article in the December 24th Journal of the American Medical Association describes the role of the artificial kidney in saving the life of a 45-year-old woman suffering severe congestive heart failure.

It marks another triumph for the mechanical kidney, originally designed to substitute for malfunctioning kidneys and later found to be useful in treating poisoning victims. The artificial kidney, which duplicates the work of the kidney by filtering waste products from the blood, can remove poisons from the body much faster than the human kidney.

Drs. William M. Lemmon, Teruo Hirose, Robert A. O'Connor, and Charles P. Bailey, New York Medical College, Metropolitan Hospital Center, said the "intentional use of the artificial kidney for the treatment of congestive cardiac failure, per se, has not been described previously."

Congestive heart failure is a disorder of the heart's function characterized by the accumulation of blood and fluids at various sites in the body. Surgical intervention is not possible unless the excess water can be removed. While this can be accomplished with most patients by using normal procedures, occasionally there are patients who do not respond. Death "not infrequently" is the inevitable result among these patients.

The patient in the case reported fell into this category. The failure to remove the excess fluid prevented an operation to correct her heart defect (mitral stenosis). It was then decided to try the artificial kidney. They found that several pounds of fluid could be removed through use of the device and that after its use the patient became responsive to previously ineffective measures.

Following the success of this treatment, an operation was performed and the patient "was cured completely."

Alcoholism . . . .

JAMES ASA SHIELD, M.D.

The following article is the first of a series of articles to be written by the members of the Committee on Alcoholism. This committee consists of Dr. William S. Sloan, Chairman, Dr. Ebbe C. Hoff, Dr. William F. Gibbs, Dr. James Asa Shield, Dr. John J. Jofko.

Office Treatment of the Alcoholic

Do-It-Yourself Medicine-Alcohol

The trend toward "do-it-yourself" is nothing new for the alcoholic. He has long been treating his personality problem by dosing himself with alcohol. Just as when the home do-it-yourself fan paints himself into the corner and, finally, has to call in a professional painter, so the alcoholic, figuratively speaking, finds himself in such a position that eventually he, too, seeks professional help.

When the alcoholic presents himself for treatment, it is imperative that we as doctors accept him as a sick person suffering from a serious, debilitating disease which interferes with his functioning capacity.

His alcoholism must be considered to be only a small part of the personality disorder he presents. Alcoholic addiction is treatable, but it is a medical problem of increasing frequency, and should be viewed with alarm by industry and the general public.

Often, in the first interview we learn that the alcoholic began drinking in college or in that age group. Probably, he was a little ill at ease at parties or gatherings, and after the first drink he became more comfortable. So, he begins to "self-medicate" himself with alcohol, and at this age, we find him drinking a little more than the other boys and getting a little drunker more frequently.

In this first interview and in subsequent interviews, he does not tell you all. He himself does not understand all about the sociological, the psychological, and biological reasons for his addiction. Furthermore, he does not understand how the culture of his family backround, the actions, and the reactions within his family revolving around him towards others, and others towards him have contributed to his feeling of being accepted, loved, rejected, or just put up with.

The therapy requires understanding of the patient as he has evolved through his life experiences: the culture into which he was born, the people with whom he has lived, loved, and by whom he has been loved or rejected have much to do with his present emotions. The alcoholic's illness is almost certainly emotionally motivated. His emotional reactions have affected his past adjustments and his present feelings about the society through which he has evolved, and is living with at present.

When we say that the physician must treat the whole individual and not just his disease, it is not just a trite phase. It is a reality that the physician today must accept. This demands that we approach the complaint of alcoholism as merely a symptom, and turn to the basic disorder and study it in a comprehensive manner; physically, calling on all the biological medical aids; psychologically, making use of testing techniques, such as the Rorschach and other personality tests, that help in the appraisal of the patient's psychological reactions, and studying the patient's adjustment from year to year, and making use of any other sociological background data that is available through interviews with the patient, his family, and other friends.

Cancer Trends....

ARNOLD M. SALZBERG, M.D.

Malignant Neoplasms of Infancy and Childhood in Virginia. I. Incidence by Mortality

It is proposed to evaluate cancer in children in Virginia in a series of brief communications in order to ascertain the role of this disease in the child-health complex on a local level.

Mortality statistics as a guide to the incidence of malignant neoplasms, including those of lymphatic and hematopoietic tissues, will obviously under-estimate the true incidence for a variety of reasons. Nevertheless, this approach can furnish some insight into its prevalence.[3]

The relative importance of cancer as a cause of death in infants and children has increased with the decline in death rates from more preventable and curable illnesses. In addition, a certain percentage of lives so prolonged develop cancer so that an actual increase may also have occurred.[1,3]

In Virginia, in 1958 and 1959, cancer ranked fourth as a cause of death in the one through four year age group. It accounted for 65 patients, or 8% of the total deaths and was exceeded by accidents, influenza-pneumonia and congenital malformations. Between five and fourteen years, malignant neoplasms are the second most frequent cause of death, surpassed only by accidents. (Table I) These orders of frequency in both age groups are duplicated on a national level.[4]

In 1957 and 1958, 163 infants and children from one through fourteen died of cancer, including leukemia, in this state. This accounted for 10% of the total deaths in this age group. (Table II) Again, this agrees rather well with the national death rate of 9% due to malignant neoplasms in this age period.[1]

TABLE I

MORTALITY ACCORDING TO AGE OF FOUR LEADING CAUSES OF DEATH IN VIRGINIA, 1958 AND 1959

Rank	Age Group and Cause of Death	Number	%
	1-4 Years		
	All Causes	850	100
1	Accidents	237	28
2	Influenza and Pneumonia	137	16
3	Congenital Malformations	95	11
4	Malignant Neoplasms	65	8
	Other Causes	316	37
	5-14 Years		
	All Causes	713	100
1	Accidents	263	37
2	Malignant Neoplasms	91	13
3	Congenital Malformations	50	7
4	Influenza and Pneumonia	37	5
	Other Causes	272	38

TABLE II

NUMBER OF DEATHS FROM MALIGNANT NEOPLASMS IN CHILDREN COMPARED TO TOTAL DEATHS IN CHILDREN FROM ALL CAUSES IN VIRGINIA, 1957 AND 1958

Age Group	Number of Deaths		% Due to Malignant Neoplasms
	All Causes	Malignant Neoplasms	
1-4	853	67	8%
5-9	395	53	13%
10-14	314	43	14%
Total	1,562	163	10%

There were 8902 deaths from cancer in all age groups in 1957 and 1958, of which 172 or slightly below 2% occurred between birth through fourteen years. (Table III) This is higher than the 1.4% reported from New York City in 1957.[2]

It is apparent that childhood cancer com-

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prises a small segment of the total cancer problem, but a reasonably large segment of the mortality within its own age group.

TABLE III

MORTALITY FROM CHILDHOOD CANCER AS A PERCENTAGE OF TOTAL DEATHS FROM CANCER IN VIRGINIA, 1957 AND 1958

AGE GROUPS	DEATHS FROM CANCER	
	No.	%
All Ages	8,902	100
0-4	76	
5-9	53	
10-14	43	
Total Under 15 years of Age	172	2

The assistance of Mr. Deane Huxtable, State Registrar, and Miss Mary Boozer, Statistician, Virginia Bureau of Vital Statistics, is gratefully acknowledged.

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Heart Month

Once again February, the Month of Hearts, is here. For the people of Virginia and throughout the nation, this month brings to mind two heart symbols, the Valentine and the Heart and Torch emblem of the Heart Fund Campaign.

During the entire month of February, the Heart Fund Campaign is in progress. At this time, the public is being asked to contribute to the support of the programs of heart research, education, and community service which are supported by the Heart Fund. Virginia Heart Association, and its chapters and councils, which are sponsoring the campaign in Virginia, have set a combined goal of $415,000.

Part of the funds raised in the Virginia campaign will be used to continue and expand heart research which is under way at the Medical College of Virginia and at the University of Virginia School of Medicine, and to assist with heart research sponsored by American Heart Association. Also, a portion of the donations to the Heart Fund will be used to develop further the programs of heart education and various community services throughout Virginia.

Contributions may be sent to the Virginia Heart Association, 812 West Franklin St., Richmond 20, Va., to the local Heart chapter, or to "Heart," c/o Local Postmaster.

Mental Health . . . .

JULIAN G. HANLON

Social Service Responsibilities in a State Hospital

This is a broad topic which has been assigned to me today, and it seems that I have given this talk many times before in many different places. Each time my paper has had to be rewritten because the responsibilities of social service, like every other department in a state hospital, are constantly changing, and it is necessary to revise one's concepts of these responsibilities from day to day and month to month.

My invitation to speak on social service responsibilities has generally come from one of two sources. On occasion I have been invited into a hospital by a Superintendent or Clinical Director who was vaguely dissatisfied with his Social Service program. My assignment then was to "jack up" the Social Service staff. Sometimes the invitation has come from the Social Work Department which felt that it wasn't "understood or appreciated." My assignment here was viewed as that of a special pleader or interpreter for the Social Work group. I do not regard my appearance here as serving either of these purposes, since I have been invited both by the Superintendent and the Chief of Social Service. I will, therefore, try to take a position somewhere between these two extremes and, hopefully, make a contribution in both areas.

Actually, I believe that most hospital Social Work departments do need "jacking up", or, to put it into more professional terms, critically evaluated, now and then. In a large institution such as a state hospital, with its terrific pressures on everyone and where there is always more work than a Social Work department can handle, it is very easy to feel justified by busyness. In so doing, a department excuses itself from ever taking a hard look at its on-going social work practice or the goals which the department is striving towards.

There is also something to the idea that social work is not adequately understood or valued by other disciplines in the hospital community. Please note that I deliberately omitted the word "appreciated", for I am inclined to suspect the maturity of any department that yearns too desperately for appreciation. Appreciation comes following performance rather than before. I realize also that this problem of more complete understanding is not the problem of Social Service alone. One can hear the same plaint from clinical psychologists, from nurses, from therapists, occupational and physical, and, yea, even from physicians. All of these groups periodically have the feeling that life could be beautiful and therapeutic success greater if only the skills of their particular profession were fully recognized and used to the maximum.

At the root of much of this trouble, I believe, is our pious dedication to the concept of teamwork and our actual preoccupation with problems of our own discipline and the advancement of our own viewpoint which may take us afield from the best interest of the people we are supposed to be serving. We struggle for personnel to fill positions in our hospitals but do not work as hard on maximum utilization of these individuals for the benefit of patients. In a sense we have a tendency to take our eye off the patient, and if any of you are golfers and

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I may draw a parallel, you are well aware that this can lead to trouble and poor results.

And now back to social work! It wasn't too long ago that as I went my round from state to state as a Regional or Area Consultant, I seemed to be waging warfare on the concept of the social worker as an odd-job person. If the patient lost his baggage, then, naturally, Social Service was to look for it. If it was decided that a bulky, twenty-year-old medical record be abstracted, then the social worker did this. Letter writing for illiterate patients was also appropriate utilization of social work time. Many social workers who had invested heavily in professional education, equipped with hard-earned Master's Degrees and progressive field experience which had brought them finally into psychiatric social work, had the feeling that their contribution to patient care and rehabilitation could be on a higher level. I recall that gradually some of these routine tasks began to be sloughed off, and one saw the social worker emerging in the state hospital as a professional person. The social worker began to appear at staff meetings, not just to copy down the diagnosis which was then reported back to the department. She began to participate in staffing, and through her contribution helped to delineate the patient's difficulty and contributed to the formulation of a diagnosis and treatment plan. She began to be heard with increasing respect when discharge plans were formulated. At this point all of this descriptive development sounds fine, laudable, and, above all, reasonable. When and where this happened it was all of these. But this was spotty and ever so gradual. I suspect that at certain hospitals many social workers are still performing in the old way, due, to a great extent, to their own lack of clarity as to their major role within the hospital as well as the inability of certain administrators and medical staff to see or accept them in any other way.

In some areas this change was even more dramatic. I have heard this age not so facetiously referred to in social work circles as the "age of seduction". Social workers began to work more directly with patients on a face-to-face, one-to-one basis. Since social workers in their training had for years been prepared through social case work techniques to operate in this way, this should not have been a surprising development. However, it was not without its problems, some of which still exist with us today.

During the "age of seduction" in social work we became enchanted by the opportunity to work so closely with psychiatry. The social work field moved very rapidly towards becoming "psychiatric". The attachment of a psychiatric label to the social worker became a symbol of prestige, and I think deservedly so. On the other hand, it was not uncommon for many social workers to add this psychiatric title merely because they were filling jobs in a psychiatric setting rather than because of any special training which gave them a right to style themselves in this fashion. I believe it was about this time that one began to hear the words "junior psychiatrists" applied to social workers somewhat derisively or else used by responsible social workers in affirming the separateness of social work and in protesting that "junior psychiatry" was not the goal of their profession. There was some concern expressed in the ranks of psychiatry over social workers' aggressiveness, although, if my psychology friends will permit, I do not believe they were considered as threatening as clinical psychology by our psychiatric colleagues. There was a feeling that basically we "knew our place" in the hierarchy.

On the other hand, it is true that we found social workers on occasion taking over the title of therapists, with the blessing of many psychiatrists, I might add, but also with some misgivings on the part of other social workers. My impression is that this did not happen with any frequency in state hospitals but was more than likely confined to psychiatric out-patient clinics. This was due, I suppose, to the fact that the level of social work training in state hospital staffs at that time was considerably lower, but an

even more important factor, in my opinion, was that the realities of life in a state hospital and the pressures of the job did not permit the luxury of "intensive therapy" for social workers or anyone else on the hospital staff.

One must admit that many of these individuals were, and still are, skilled therapists, able to understand and utilize teaching of their psychiatric mentors and even contribute something to the psychiatrist's understanding. They were able to use themselves with discipline in a helping relationship with patients and perfectly capable of providing help to disturbed people.

I have skimmed lightly over this historical development because I did not intend it to be my major theme, nor do I wish to give the impression that these days are completely behind us. I do believe, however, that today we have a greater development of the social worker's role in relation to the psychiatric patient and considerably more clarity as to the areas in which the social worker operates most effectively. Much of this clarity has come out of the work of social service departments in large federal and state hospitals. I think this was because in these hospitals one was able to see all the complexities of a patient's problem and one was forced to grapple with that large area of patients' needs beyond "therapy" in a narrow sense. I am convinced that no matter how jaundiced or prejudiced one feels about the concept of teamwork, a tour of duty in a state hospital impresses upon one's mind that a therapeutic program for patients cannot be the province of any one professional group and that a good job cannot be done without teamwork in its finest sense. It is in this kind of setting, where many dedicated people were groping for answers, that psychiatric social work, I believe, has been able to plant its feet most firmly on the ground. I cannot imagine any place that would be a greater challenge to a social worker or where there is a greater opportunity for effective service than in a large hospital such as this.

I would like to comment briefly on the social work role at various points in the patient's hospital life, in the hope that these remarks may stimulate comments, or at least some self-examination as to the way things are at present here at Eastern State.

Pre-admission. At one time, if you mentioned pre-admission social service in the state hospital, your suggestions would be waived aside as being visionary or at least of dubious value. I no longer find this to be true. Many hospital social service departments are moving out to meet the patient and his family in the community before hospitalization is necessary. I think this has several purposes, all of them sound. First of all, it gives an opportunity for better interpretation of the hospital, its functions, and its limitations, which, I believe, may bring about more appropriate admissions, minimize the number of emergency admissions, and smooth the way for easier admissions where this is indicated. In many cases through this service it is possible for the patient to participate in his own hospital admission. It certainly provides an excellent opportunity for establishing a beginning relationship with the family which will enable them to maintain a positive, non-rejecting attitude throughout the period of care and ultimately help them to receive the patient back with more understanding and readiness. Such services have proved themselves extremely helpful in institutions for the retarded, where the specter of a long waiting list hangs over the hospital staff and forces them to work with a sense of urgency because of the real and fancied pressure from the community. I think we need to examine this phase of social service work more closely, and I would like to see some small descriptive research projects which would outline some of the problems and strengths when one adds this to the social service program.

Admission. Second, and in logical sequence, is the time of intake. Hospitals have made much progress in this area, I believe, through humanizing their admission procedures, providing early workups of patients, and moving them quickly into treatment programs where this is indicated. For the

social worker, early work with the family at this point is quite important, and by this I do not mean merely grabbing the harassed relative and extracting history information from them at admission. It seems to me it might be far sounder if we could devote our time to more sympathetic attention to the problem the relative may have in hospitalizing the patient. Some awareness that this relative's life may be dislocated at this point and that the problems he faces after he leaves the patient may be more burdensome than any the patient will have to grapple with for the next few months. Our treatment of the relative at this point sets the tone for what he may expect from the hospital in the future. I see this as an area requiring extremely skillful handling. I feel certain that it pays dividends at later periods.

I cannot leave this area without a few words about social histories. I confess to somewhat unreasonable personal feeling about this, since as a younger social worker I did many social histories which involved slavish adherence to a very complete life history outline, much of which, as I look back, was not used by anyone for the patient's benefit. I like to think, then, that histories done by the social worker, or secured by them from community agencies, will be more than factual information on a piece of paper. History taking should be considered part of the treatment process and calls for great skill and sensitivity on the part of a well-trained social worker. The product should be a clear delineation of the patient's difficulty in his social setting, the circumstances which led to his hospitalization, and the positive and negative factors in his life which will have to be considered as the hospital plans with him for his future. I think we have actually moved far away from the day when social workers routinely did these studies on every patient, only to find that the patient had been discharged when the material was written up, an indication of how little importance was attached to the exhaustive life history by the physician.

Social Service During Hospital Care. What is the social worker's role within the hospital and before a clear-cut plan for discharge is set up? Somehow it seems to me that once a patient is admitted, and particularly where longer term, continued treatment is agreed upon, the patient may well fall into a void as far as social service is concerned. Social workers move on to become involved with new cases and may not hear much about the patient until months later when discharge is thought about.

This may have been all right when turnover in our hospitals was at a minimum, but I question whether it is a satisfactory situation today. More patients are leaving the hospitals and we know they are not all well when they leave. Has there been continuous work with the family during this period to correct those factors in the environment which may have contributed to hospitalization? Is it recognized that the patient, even while under treatment for his illness, might be able to work with the social worker, planning in the healthy area of his life? I think this can work out if there is professional respect between physician and social worker and sufficient clarity as to both their roles to enable them to work together for the patient's interest. For most patients discharge planning should start, if not at admission, at least as close to it as is psychiatrically sound. Relatives must be kept related to the patient's progress or lack of it. Many disciplines within the hospital can and should work directly with the patient without infringing on the medical responsibility of the physician, but at the same time with full knowledge and awareness of the hospital's goal for this particular patient. There are practical tangible things that families must do when a patient goes away or is preparing to come home. The wife may ask, "Should we move into a smaller house if my husband is to be away for six months or for years?" "Should I seek employment?" I think the hospital, through its social service department, has the responsibility to be close enough to these relatives to give them available and necessary information or to see that

it is made available to them through interested and informed community agencies. On continued treatment, then, there is a place for the social worker. It may not involve major responsibility for the patient, but it does mean an awareness of the patient's progress and constant availability to make the proper social work contribution whenever and wherever this is indicated.

Discharge. I have touched briefly on pretrial visit planning already, but I would like to speak to this point directly. Over the years hospitals have been criticized for what appears to outsiders as haphazard, careless discharge policies. Much of this criticism is unfounded, but this does not free us from examining our ways of working to see where we might be failing.

At one time the pet peeve of many outside the hospital was the impulsive discharge on visiting day which appeared to ignore planning for the patient already under way or failed to take into account that discharge may involve more than the patient's readiness. I do not think all these releases were bad, but I wonder how many readmissions spring from failure to properly assay the community's and the family's readiness to receive the patient back and to offer him a stable environment where he has a reasonable chance of adjusting successfully.

The answer to these complaints has quite often been a haughty "We must discharge when the patient can no longer benefit from hospitalization. We cannot wait for the perfect moment or for the slow wheels of welfare or other community agencies to start." "The community cannot dictate who comes and goes to the hospitals", and "The fault lies in the community's unwillingness to receive our patients back once they have maneuvered us into admitting them."

I do not believe the community wants to dictate hospital discharges, and I suspect that when the hospital takes the time to explain its reasoning, most communities are understanding and acceptant. But you do owe them that explanation! It is all right to have doubts as to a patient's chances for adjustment following release. It is not all right to base discharge planning solely on the patient's adjustment to the hospital with no knowledge of the factors he will have for him and against him when he leaves the hospital environment.

The social work department with its lines into the community should be prominent in drawing this picture for the staff which jointly must make these decisions.

After-Care. This is the area of our work which seems to be receiving the greatest amount of attention today. We attribute many things to the influence of new drugs, but I think we are safe in saying that they have played a prominent part in forcing us to think about what happens to the patient who no longer needs hospitalization or who can manage even marginally in the community following a stay in the hospital. Interest in this area is certainly interdisciplinary, and, as a well known comedian used to say, "Everybody wants to get into the act." Thus we find increased interest on the part of rehabilitation counsellors, public health nurses, welfare departments, and, what is probably most welcome, the private physician, who is more and more involving himself with ex-hospital patients. The out-patient clinics, many of whom have successfully avoided these patients for years, are being pressed to include them as recipients of their service. At this hospital you have had for a number of years your own follow-up clinics, and Western State is about to embark on a follow-up program supported by a federal grant which will include group therapy sessions prior to and after release in the patient's home community and a resident social worker in the Northern Virginia area to serve as liaison with community agencies on behalf of hospital patients.

In the area of after-care, I suppose the community really asks the impossible of us. In effect they say, "If you are going to release patients, it is up to you to see that they are supervised and helped or, in its most negative form, to see that they don't cause any trouble."

Our problem, then, boils down to the question as to whether we can develop a system where we can be comfortable about our released patients, a system that would give us a little more assurance as to the patient's later adjustment and less crossing of our fingers as we apprehensively release them without clearly defined plans for giving them the help they may require.

I think this is more possible than our current success would indicate. Undoubtedly we have to first reach the point where every readmission is not regarded as an index of failure, particularly if, when the patient was discharged, a plan for his placement in the community was carefully thought through rather than haphazardly backed into. I pose these questions for you: Where drugs are necessary for a patient, has a plan been worked out which is clear to the patient, his family, and the hospital as to how he will receive his drugs after the initial supply has been exhausted? Has the family physician, who once was interested in the patient, been invited to involve himself again in his after-care, with adequate knowledge of the course of his hospital treatment? Where some kind of continuing support, either psychological or financial, is indicated for a patient, have you succeeded in finding a community resource which will cover the patient's need? How many patients have been placed in foster homes, or have you managed to push aside such a program as being too expensive in time and money for only a small group of patients? When you do place patients in foster homes, is it a question of merely finding a place for them to live or is the placement viewed by the patient, his family, and the caretaker as a continuation of hospital treatment? Are you so conscious of the joy of decreasing patient loads that you rush patients into the community without supervision or help? Are you placing (I almost said "railroading") patients into poor nursing homes and other questionable facilities so that their second lot may not be any improvement over prolonged hospital stay.

Let me not give the impression that after-care is the exclusive responsibility of social service. It certainly is not. I do know, however, that if the social service program is weak, inadequate, and has a limited horizon, an after-care program will certainly fall down. A hospital should be able to count on the social worker to develop a network of community facilities, fully aware of the hospital's mission and ready to do their part in needed service to hospital patients where this is appropriate. This means social workers who think in terms of broad community planning and responsibility and it also means something as narrow as having a car available when the social worker needs to go outside the hospital walls. It means social workers who are imaginative, not stodgy, who are confident that their social work skills are needed if the hospital is doing a complete job. Along with this confidence goes a real respect for the contribution of everyone, from the gatekeeper to the superintendent, and a conviction that teamwork can be more than a cliche, bandied about by visiting lecturers.

Pre-Paid Medical Care....

Edited by
RICHARD J. ACKART, M.D.

Medicine and Blue Shield

The A.M.A. Clinical Meeting held in Washington, D. C., November 28 through December 1, may ultimately prove to have influenced the future of Blue Shield more profoundly than any other single event in the history of the Plans. A resolution adopted by the House of Delegates specifically committed the A.M.A. to take the initiative in organizing and implementing a program of effective action to stimulate ". . . maximum development of the voluntary non-profit prepayment concept to provide health care for the American people."

To establish the national organization required to accomplish this goal, the A.M.A. resolution calls for participation by the American Hospital Association, the Blue Cross Association, the National Association of Blue Shield Plans, and the American Medical Association in developing a basis for joint action aimed at overcoming the obstacles standing in the way of extending the scope of Blue Cross and Blue Shield to meet maximum public need.

The text of the A.M.A. resolution on prepayment Plan development follows:

> "Whereas, It has been widely recognized that voluntary health insurance is the primary alternative to a compulsory governmental program: and
>
> "Whereas, The public has shown its confidence in this voluntary system; and
>
> "Whereas, Current social, political and economic developments compel a new and revitalized effort to make voluntary health insurance successful; and
>
> "Whereas, The American Medical Association has consistently pledged itself to make available the highest type of medical care; therefore be it
>
> "*Resolved*, That the House of Delegates direct the Board of Trustees and the Council on Medical Service to assume immediately the leadership in consolidating the efforts of the American Medical Association with those of the National Association of Blue Shield Plans, the American Hospital Association and the Blue Cross Association into maximum development of the voluntary non-profit prepayment concept to provide health care for the American people; and be it further
>
> "*Resolved*, That similar leadership be undertaken to coordinate the efforts of private insurance carriers through conference with their national organizations; and be it further
>
> "*Resolved*, That, where feasible, efforts be made to cooperate with representatives of other types of medical care plans, other professional groups, and representatives of industry, labor and the public at large."

Commenting editorially on this resolution, the Washington (D.C.) *Evening Star* observed that the physicians who ". . . stimulated the A.M.A. action and who are working toward its fulfillment, harbor no illusions that any single new plan can be developed or even proposed which would satisfy all the various medical care needs which currently are being discussed. But they do believe that drastic improvements can be made in existing voluntary Blue Cross and Blue Shield plans, in coordination later with commercial prepayment programs, which would decrease the need for government interven-

tion; and this is the goal toward which they are working."

Concluding, The *Evening Star* observed that "These efforts . . . can produce nothing but good. The medical voluntary prepayment movement since World War II has made phenomenal advances, and that fact too often is obscured by criticism of its deficiencies. What is important now is that these shortcomings are being given the attention they deserve. . . ."

In a report accepted by the House of Delegates of the A.M.A., the following observations concerning the present and the future of Blue Shield were made:

"The medically sponsored Blue Shield Plans, which now provide coverage for medical care benefits to more than 45 million persons, represent the most convincing evidence available of medicine's capacity to meet the nation's health needs through private initiative. Indeed, through the physician sponsored Blue Shield program, our profession has demonstrated its capacity to provide both the leadership and enlightenment necessary to solve the economic problem of providing health care without sacrificing the individual and professional freedoms on which high quality care depends."

"It is not possible to discuss the future of Blue Shield without reference to the principal resource the Blue Shield Plans have available to apply in shaping their future. This resource is the medical profession which has been responsible for the organization of Blue Shield. The doctors of the nation alone hold the key to Blue Shield's future, and their leadership, or lack of it, will inevitably determine whether the Plans are prepared to respond to the opportunities that lie ahead."

Here in Virginia The Medical Society of Virginia has accepted the responsibility of leading Blue Shield toward even more effective service to the people of the State. In this effort the Society will need the suggestions and the cooperation of each and everyone of its members.

Aids Mild Diabetics

A drug designed to fight mental depressions, imipramine hydrochloride, has been found to benefit persons with mild diabetes. Results obtained in five patients are reported by four Cincinnati physicians in a preliminary communication in the October 1st Journal of the American Medical Association.

The effect of the drug was noted when it was prescribed for several mild diabetics who were also depressed. Administration of the drug was followed by a decrease in the amount of sugar excreted. A high level of sugar excretion is a symptom of diabetes, which is a disorder that prevents the body from utilizing sugar normally.

Each subject's response to the drug and to discontinuance of it was tested several times. However, it was stressed that their findings were not conclusive because of the small number of patients studied.

Further investigation is warranted because it is important to study any agent that demonstrates an effect on the clinical course of diabetes.

The authors are Drs. Stanley M. Kaplan, James W. Maas, John M. Pixley, and W. Donald Ross, who was associated with the University of Cincinnati College of Medicine and the Cincinnati General Hospital.

Current Currents

WHITE HOUSE CONFERENCE: The big news, as this issue goes to press, is the White House Conference on Aging which has just concluded with the recommendation for a Social Security health care plan similar to the one favored by President Kennedy. The Income Maintenance Section, which dealt with this issue, voted in favor of the recommendation 170 to 99. A minority report was released which declared that incorporating medical insurance under Title II of the Social Security Act "would interfere with the physician-patient relationship; that it is unnecessary because of the potential growth of voluntary insurance, and that all needy aged can be cared for by public assistance through the recently enacted Federal program of health care for the low income aged." The minority report pointed out that the majority report was not in accord with the official expression of the majority of state conferences on aging.

Most observers agree that the Kennedy administration will use the report of the Income Maintenance Section to further the compulsory social security approach to health care of the aged, notwithstanding the results of the state conferences or the minority report. AMA points out that while the results of the Conference were not totally unexpected, they do not presage certain passage of the compulsory social security approach to health care of the aged in the 87th Congress.

KENNEDY BILL: The new so-called Kennedy bill will most likely have been introduced by the time you read this issue of Current Currents. Advance reports indicate that it will be limited to hospital and nursing home care. It will probably not include physicians. The bill is expected to cover some 14 million persons. It will provide for anyone who receives OASDI benefits, including 2½ million persons under 65, notably children of beneficiaries.

HR 10: Senator Eugene J. Keogh (D., N.Y.), has introduced HR 10, which like its predecessor of the 86th Congress, provides a retirement program for the self-employed. A similar bill, S.59, has been introduced in the Senate by Florida Democrat George A. Smathers.

HR 10 would amend the pension plan provisions of the Internal Revenue Code by adding a two step requirement for all plans and special requirements for the self-employed. If an employer has three or more workers, they must be covered and have a vested interest in the plan. The self-employed would be able to deduct ten percent of income or $2,500, whichever is smaller, from the self-employment earnings of the

business for which the plan would be established. No benefits would be paid before age 59½, unless there is death or disability, but would have to begin at age 71.

PHYSICIAN CONTRIBUTION TO FREE CARE: AMA has reported that United States physicians are annually providing free medical care amounting to 658 million dollars. These figures were obtained from a survey conducted by the magazine NEW MEDICAL MATERIA. It was brought out that more than 98 percent of our physicians give free medical care, and that 60 percent contribute 10 percent or more of their working hours to this free service.

NEW BOOKLET: Members will wish to make good use of a new illustrated booklet just published by the American Medical Association. Its title is "The Cost of Medical Care" and it contains information everyone should have. Did you know, for example, that the average American spends 6¢ of his consumer dollar for health? This compares with 27¢ for housing, 22¢ for food, 10¢ for clothing, 12¢ for travel, 6¢ for recreation, 5¢ for tobacco and liquor and 12¢ for all other items.

The booklet also reports the spectacular progress made by medical science during the last 20 years. A child born today can expect to live 10 years longer than one born 20 years ago; new antibiotic drugs prevent pneumonia that used to kill one in every three or four persons it attacked. Low cost vaccines now offer protection against many serious illnesses; the danger of infection through surgery has been greatly reduced; today's average stay in the hospital for an appendectomy is four days, compared with fourteen days 20 years ago.

The booklet reports that physicians average sixty hours of work each week and see more patients than 20 years ago. Their fees, however, have gone up much less than the average price of all "consumer goods and services".

Copies of this booklet may be obtained by contacting the State Office of The Medical Society of Virginia.

A SPECIAL SALUTE: Dr. Vincent W. Archer, Past President of The Medical Society of Virginia and currently a member of the AMA House of Delegates, has been designated the recipient of the Gold Medal of the American College of Radiology. The presentation will be made during special ceremonies at the Drake Hotel, Chicago, on February 10. The Medal is being awarded "for distinguished and extraordinary service to the American College of Radiology and the profession for which it stands".

DID YOU KNOW? According to Insurance Economics Surveys, the average American consults his physician approximately five times a year. This American can also choose between some 1,200 organizations currently providing health insurance for the public.

Public Health....

MACK I. SHANHOLTZ, M.D.
State Health Commissioner of Virginia

Sylvatic Rabies Control in Virginia

Every year hundreds of persons in Virginia receive anti-rabies treatment because of contact with or exposure to animals which have been proved to be rabid or which have escaped capture and there is doubt as to freedom from infection. In many instances the contact or exposure has not been such that infection of the individual has taken place. However, it is a fact that once developed, there is no treatment for rabies; prevention is the necessary measure. It happens often that the animal is not available for examination or the history is not sufficiently clear to determine the intimacy of contact or exposure. In such cases there is but one decision—treat the human being, do not take chances with his life.

Rabies in animals is a continuing problem in Virginia and involves particularly the entire western border of the State and, to a lesser degree, its northern border. While there has been an annual decrease in the total number of positive animals during the past five years, the incidence of rabies in wildlife still causes concern.

TABLE I

YEAR	TOTAL	HEADS POSITIVE			
		IN WILDLIFE		IN DOGS	
		No.	%	No.	%
1955	373	208	55.8	101	27.1
1956	299	142	47.5	64	21.4
1957	318	232	73.0	21	6.6
1958	252	153	60.7	29	11.5
1959	168	102	60.7	25	14.9

Table I shows the number of heads positive for rabies by year for the past five years together with the number and percentage referable to wildlife and to dogs each of these years.

Successful dog vaccination programs undoubtedly have played the most important role in reducing the incidence of rabies in the canine population. A build-up of populations, combined with a high natural susceptibility of foxes to the disease, plays a part in the rise in incidence of rabies among animals in wildlife. This changing picture is nationwide as is evidenced by the fact that during the past ten years (1948-58) the number of reported cases of rabies in wild animals has doubled.

Although the number of positive dog heads may approach closely the actual number of cases in the canine population, this is not true in other species. In wildlife it is the animal that comes to the attention of man—the suspicious animal, the one which is acting abnormally, or the attacking animal—which is destroyed and examined. Those sick and dying in their natural habitat are not discovered. It is estimated that only 1% of the actual number of cases of rabies which occur in predatory animals in the forests are brought to laboratories for examination.

The occurrence of rabies in his domestic livestock is of vital concern to the farmer. During the past five years (1955-59) 274 farm animals have been diagnosed as infected with rabies. One hundred and fifty of these, 54.7%, have been in the 18 counties west of Roanoke. Here again, the number of positive heads reported is a poor indication of the actual number of cases. The cow's head is large and difficult to handle. If several animals present the same chain of symptoms, the head of one which has died is examined and it is presumed that the others were infected with the same disease. A conservative estimate would be that for every head of a horse, cow, or sheep which is examined and found positive, four other cases exist but remain unexamined and unreported for various reasons. Furthermore, unless there has been a human exposure, the veterinarian's clinical diagnosis (in cases seen by a veteri-

narian) is usually accepted by the owner and the animal is destroyed before human exposure can take place.

Sylvatic rabies epizootics seem to follow a geographic pattern: the disease moves into a county from an adjacent county where an epizootic has been prevailing. There appears to be a population threshold which, when exceeded, provides a sufficient number of animals to sustain the virus and an epizootic results. There may be a yet undiscovered synergistic action between rabies and another disease and the susceptibility to rabies of a certain animal may be affected by the presence of this second disease. Conceivably, the season of the year or the temperature may influence the range of the susceptible animals or may effect their food supplies. The virulence of the virus may play a part in determining the final population necessary for the development of an epizootic state. While the flexibility and intangibility of this population threshold are recognized, there are indications that such a threshold exists.

Following the occurrence of an epizootic of rabies in a county, there is a period during which few cases are encountered. There is no clear-cut reason for this sudden drop in cases but we believe that reduction of the susceptible population is the chief cause. The disease is a fatal one and kills out those animals that acquire it. It may be that the entire susceptible population does not meet the virus because, before this can happen, it is carried out of the area by means of one of the factors mentioned above. Reports show that year after year sylvatic rabies moves from county to county in southwestern Virginia.

Trapping of predatory animals in areas where there are indications that their populations are high, has demonstrated that the spread of rabies can be controlled in wildlife.

The General Assembly of 1954, recognizing the need for control of rabies, adopted a section (29-213.1) providing that "whenever the State Health Commissioner is informed that an outbreak of rabies has occurred in a county or city, he may, after consulting with the Commissioner of Agriculture and Immigration and the Executive Director of the Commission of Game and Inland Fisheries, adopt a regulation declaring the existence of rabies in such a county or city and containing such requirements as are hereinafter set forth. Such regulations shall be prominently displayed throughout the county or city and shall be published therein by signs or otherwise call attention of the public to the existence of such an outbreak."

Under provisions of the same General Assembly, the Commission of Game and Inland Fisheries was directed to "employ and make available to local governing bodies on request, a person skilled in the control of wild animals capable of spreading rabies."

The procedure has been for the State Commissioner of Health, on hearing of an increase in incidence of rabies in wild animals in any area, to request the Executive Director of the Commission of Game and Inland Fisheries to institute measures to trap predatory animals in that area. This has been followed by consultation on the part of the "person skilled in the control of wild animals capable of spreading rabies" with the Board of Supervisors of such a county and an offer by him to train individuals they might designate in the art of trapping and to purchase the needed traps at the expense of the county. It is needless to say that persons trained in such a way are not as skillful as those who are constantly trapping. Another factor has been that initiation of such control has come from outside the county.

In June, 1960, the Public Health Veterinarian proposed to the Commissioner of Health that full-time trappers be employed and that these men be made available to any area upon request of the governing body of the area. These trappers would function in the area until a wildlife census, conducted by their supervisor, indicated that the number of animals concerned was likely to be below the population necessary to sustain

rabies in such animals. This would be confirmed by a reduction in the number of rabies cases reported.

The use of full-time trappers has been carried on in several States. The proposal for their use in Virginia is slightly different. In other States the trappers ordinarily go into an area *after* the epizootic has developed to prevent further spread among wildlife and to reduce the risk of exposure of the livestock of the area. In Virginia the Health Director of a county would note that rabies in wildlife has prevailed in an adjoining county. He would then report this fact to his Board of Supervisors and would urge them to request that trappers be sent in to reduce the population of the predatory animals of the county *before* the introduction of rabies therein. This would be a control measure to prevent the establishment of rabies within the county and thereby prevent danger of exposure of humans and loss of livestock.

The Commissioner of Health requested a meeting with the Commissioner of Agriculture and the Executive Director of the Commission of Game and Inland Fisheries. The Public Health Veterinarian of the Department of Health, the State Veterinarian of the Department of Agriculture, and the Assistant District Agent of the U. S. Fish and Wildlife Service, Predator and Rodent Control Branch, were also present. This program was unanimously adopted and each department agreed to furnish one-fourth of the cost of carrying out the program up to a certain sum, to present the program to the Governor for his approval, and to request him to make available one-fourth of the cost of the program up to a certain sum. The Governor looked favorably on the program and agreed to make the requested sum available.

The details of operation have been worked out. The position of Predator Trapper has been defined and set up in the State Personnel Office. The funds are being placed with the State Treasurer in such a manner that they can be drawn upon on authorization of the State Department of Health. The administration of the program is under the joint supervision of the Public Health Veterinarian and the Assistant District Agent, U. S. Fish and Wildlife Service, Predator and Rodent Control Branch. The predator trappers are considered employees of the State Department of Health while the Mammal Control Supervisor, under whom they work, is an employee of the Virginia Commission of Game and Inland Fisheries.

This is a beautiful example of the recognition by three of the Departments of the State of Virginia of the need to protect the lives of its inhabitants and the economy of its citizens and of their willingness to band together to work in a common cause.

This program becomes effective immediately. On being informed by the local Health Director of the imminence of a possible rabies outbreak within the county, the Board of Supervisors should request the Public Health Veterinarian, sending a copy to the Regional Health Director, to proceed with the above-outlined control measures. Because of previously described epidemiologic patterns, counties in southwestern Virginia have been designated priority areas. It is in these counties that the Health Directors are now urging their Boards of Supervisors to request initiation of this program. Two of these counties have already sent requests from their Boards of Supervisors for the predator trappers to work within their confines.

MONTHLY REPORT OF BUREAU OF COMMUNICABLE DISEASE CONTROL

	Dec. 1960	Dec. 1959	Jan.-Dec. 1960	Jan.-Dec. 1959
Brucellosis	0	0	33	27
Diphtheria	3	2	40	13
Hepatitis (infectious)	81	42	844	503
Measles	967	301	7596	15,021
Meningococcal Infections	6	10	60	88
Aseptic Meningitis	3	—	45	—
Poliomyelitis	11	6	57	290
Rabies (In Animals)	23	6	226	168
Rocky Mt. Spotted Fever	0	0	39	38
Streptococcal Infects.	538	529	6074	8450
Tularemia	5	2	38	20
Typhoid	0	2	22	27

Editorial....

War on the Staphylococcus

RECENT SURGICAL LITERATURE contains many reports of an increasing incidence of postoperative wound infection, due chiefly to staphylococcus aureus, coagulase positive. Epidemiologic explanations, some of which are based on careful bacteriologic studies and phage typing, indicate the complex nature of the problem, but fail to show any single common denominator, unless it be an increasing virulence of the organism in question. Contrary conclusions have sometimes been drawn at different institutions; in many instances these diverse opinions are entirely valid and result from environmental and personnel problems peculiar to a particular hospital. The only real and lasting protection lies in shutting off as completely as possible all avenues of bacterial contamination, while at the same time reemphasizing the proper care of the wound.

The experience of a single surgeon and his team performing all operations in the same room may prove educational. Over a two and a half year period, between 1957 and 1959, six fatal staphylococcus complications were encountered. In all six cases positive blood cultures were obtained. Three of these occurred in the year 1959. The grief and devastating effect on morale can scarcely be imagined. In five of the six cases, cardiac operations were performed, three of which were open heart procedures. The sixth operation consisted of a lobectomy for bronchiectasis. The pertinent facts concerning these cases are as follows. An anterior mediastinal hematoma undoubtedly provided a nidus for infection in one case of congenital aortic stenosis and rupture of the aortotomy occurred. In another patient, a space remaining after resection of a large ventricular aneurism allowed a collection of fluid, later shown to be infected, and rupture of the suture line followed. A ventriculotomy incision became infected in a third patient. The locus feeding a staphylococcus septicemia was not definitely established following repair of an interventricular septal defect in a patient upon whom autopsy was not performed. Staphylococcus wound infection, empyema, osteomyelitis of the sternum and toxic nephritis occurred in one patient following decortication for constrictive pericarditis. A massive wound infection and empyema, eventuating in contralateral staphylococcus pneumonia, occurred after the lobectomy. Although staphylococcus infection is not confined to any branch of surgery, it is especially hazardous in relation to cardiovascular operations.

A description of how this situation was brought under control will allow me to emphasize specific measures in the prophylaxis of postoperative staphylococcus infections. Many important features in the overall control of infection such as sterilization of routine and special equipment, general housekeeping and isolation of infected patients will be omitted

from this discussion, and only those features mentioned over which we, as surgeons, were able to exercise immediate control.

A consideration of certain unfavorable environmental conditions which might have contributed to this grievous experience focused our attention on the following:

The possibilities of heavy airborne contamination were great. Always a large number of personnel, at times totaling 15, either professional or technical, participated in or supported the open heart procedures. In a volumetric sense, because of the low ceiling, the room is small. Conversation at times was excessive. The operating room is located adjacent to a disposal room used for both clean and septic waste materials and across the hall from a busy ENT operating room. It was frequently necessary for an anesthesiologist in a supervisory capacity to float between the two operating rooms. Partly because of a centralization of supplies outside the operating room, and partly owing to legitimate activity, traffic in and out of the room has at times been alarmingly heavy. The geometry of the operating suite is such that intimate contact with the outside world is constantly renewed.

As much as possible was done to restrict sharply this traffic. Since truly effective control of traffic and significant decongestion of personnel and equipment remain a dream until more appropriate and commodious quarters are provided, and until more uniform measures are established, the decision was taken to install continuous, direct ultraviolet irradiation. The method used so successfully by Hart at Duke University Hospital was closely followed. The results seem to have justified this decision, although no illusion exists that we have acquired a panacea for all our ills. Admittedly, the donning of protective hood, glasses and eyeshield constitutes a mild nuisance to the surgeon, but these measures also serve as a reminder of the grave responsibility entrusted to him in the preservation of an aseptic environment. Limited bacteriologic studies showed an impressive reduction in bacterial fallout. Perhaps without justification I continue to harbor some apprehension about the possible ill effects of 2600 Angstrom radiation upon the tissues of the patient. Even a shallow film of saline will protect, but this has not been continuously achieved in the case of the exposed heart. Ultraviolet irradiation was also installed in the contaminated disposal room nearby, and the assurance of air sterility here was most comforting.

A recheck of the standards used for sterilization of the pump oxygenator by ethylene oxide failed to disclose any breaks in technique, and the extracorporeal circuit could not be incriminated, except possibly in one instance. Nevertheless, the conditions of assembly and transport of equipment to the operating room appeared to be suboptimal.

Constant vigilance and personal discipline must remain the key factors in the never-ending battle against the staphylococcus. All personnel with respiratory infections have been firmly excluded from the operating room. The massive inoculation of bacteria which can occur by this

route has been documented by others. In one of the fatal cases cited above a student nurse with a respiratory infection assisted at the operating table. Her infectious condition was only discovered when she voluntarily departed because of severe nasopharyngeal congestion.

Other measures adopted to reduce airborne infection included double masking and the application of clean booties over special operating room shoes. The wearing of booties appears to be the only possible solution to the universal contamination of shoes. The masking of the patient was seldom well carried out and attention has been directed to this matter. The problem of nasal carriers is too complicated and too poorly understood for discussion here. We would certainly discourage the dressing of infected wounds by any surgeon before coming to the operating room.

Mindful of the potentiality for infection tracking in along phlebotomy catheters, we have insisted on rigid aseptic precautions for the insertion of these and have withdrawn them as soon as practical following surgery.

The patient's own skin as a source of contamination has been emphasized and deemphasized by various authors. We consider the matter of sufficient importance to initiate preparation of the skin with a hexachlorophene soap at least forty-eight hours before operation. Soap continuously concentrates in suds the bacteria from the deep layers of the skin even after many minutes of scrubbing. This is a matter of great practical importance in the preparation of the surgeon's hands and arms as well as of the patient's skin. If not adequately removed by thorough rinsing, a residue of bacterial contamination remains.

We do not share the opinion which has been too frequently expressed that the hands of surgeons and nurses do not constitute an epidemiologic factor in postoperative wound infection. The importance of this source undoubtedly varies from month to month and from person to person. In a carefully conducted study Haupt cultured staphylococci from 90% of surgeons' gloves and in 14% the organisms were coagulase positive. At the end of several hours in a poorly air conditioned room, we have often found the surgeon's previously gloved hands heavily contaminated, no matter how thorough the initial scrub. The problem is magnified during the summer months because of sweating. Sweating through the sleeves of operating room apparel signals a gross break in technique which is difficult to control other than by improved air conditioning. During a long operative procedure we have encouraged a second scrub and change of mask and gown. Each surgeon should conscientiously care for the skin of his hands. The resident bacterial population in the deeper layers of the skin may change from time to time. There is less chance of invasion by pathogens in the absence of any breaks in the integument. A hexachlorophene soap or detergent should be used daily at home as well as at work. In my own experience, Gamophen soap has proved extremely kind to the skin.

An educational campaign has yielded rewarding results in reducing the

number of breaks in the glove barrier. This campaign has included an insistence upon properly fitting gloves. The use of small arterial needles held between the fingers had led to an alarming increase in the number of punctured gloves, both among surgeons and nurses. A new method of grasping even the smallest needles has been adopted. If the needle is held upside down at the junction of suture with shaft, the point of the needle is displayed well away from the fingertips and the needleholder can be easily applied.

We have found the battery-powered Weck hole detector a useful instrument. However, this method requires the surgeon to step aside and voluntarily test his gloves by dipping in a basin. We have modified the instrument to incorporate a buzzer signal. A basin connected to the circuit provides the scrub nurse with saline into which she dips bimanually each time a suture is passed. More recently the surgeons at the table have been conveniently included in this detector system, the details of which will be published elsewhere. Thus, prompt recognition of glove holes has been achieved. Until Underwriter's approval has been obtained, this system has been restricted to cases in which nonflammable anesthetics are used. A striking improvement has resulted from this battle against breaks in the glove barrier, and holes occur much less frequently.

Renewed attention has been paid certain matters of wound toilet. Thorough irrigation with saline of the pericardial and pleural cavities and the mediastinal and subcutaneous spaces has served to reduce the bacterial contamination of these areas. Among the open heart cases there have been occasional patients with a bleeding tendency at the time of closure. A special effort has been made to eliminate dead space. Appropriate tube drainage of the pericardium and anterior hilar region has aided in the prevention of sanguineous collections in the anterior mediastinum.

The results obtained by this campaign against the staphylococcus have more than justified the effort expended. Among the cases performed during the thirteen months since the adoption of these measures and among the cases protected by these measures, we have not experienced any serious staphylococcus infections. Only three small superficial, infected hematomas were observed.

The everpresent danger exists that success will lead to a relaxation of vigilance. The "surgical conscience" should not yield.

L.H.B.

News Notes....

New Members.

Since the list published in the January issue of the Monthly, the following new members have been admitted into The Medical Society of Virginia:

Charles C. Ashby, M.D., Dinwiddie
Bruce Cramer Banning, M.D., Arlington
Rogelio Menendez Borrell, M.D., Williamsburg
Robert G. Bullock, M.D., Arlington
Max Carlton Feinman, M.D., Lynchburg
Albert Andrew Fratrick, M.D., Appomattox
Herbert Claiborne Jones, Jr., M.D., Charlottesville
Reginald Paul McManus, M.D., Arlington
John G. Novak, M.D., Lynchburg
Ramon B. Perez, M.D., Wise

Local Committee on Arrangements.

For the Annual Meeting of The Medical Society of Virginia to be held in Richmond, October 8-11, 1961, the following committee on arrangements has been appointed:

Chairman—Dr. M. M. Pinckney
Hotels and Halls—Dr. William H. Harris
Scientific Exhibits—Dr. Frank M. Blanton
Technical Exhibits—Dr. Daniel D. Talley, III
Press and Publicity—Dr. William H. Higgins, Jr.
TV and Motion Pictures—Dr. Elam C. Toone, Jr.
Golf—Dr. A. L. Herring, Jr.
Entertainment—Dr. Thomas W. Murrell, Jr.
Auxiliary—Mrs. Wyndham B. Blanton, Jr.

The State Board of Medical Examiners

Held its winter meeting on November 28th at the Hotel Richmond, Richmond, with all sixteen members present. Dr. Charles J. Irvin, Roanoke, was present as a new member of the Board. The Reciprocity Committee recommended licensure of 49 doctors by endorsement of credentials from other states (reciprocity). These doctors held licensure in sixteen other states or from The National Board of Medical Examiners. These were all accepted by the Board.

The examinations were given during the next four days. The grading results of these examinations were published a few weeks after the Board meeting. These were 107 doctors who passed the examinations and received their license certificates. Of this number there were only three who were graduates of American medical schools and 104 graduates of foreign medical schools.

Dr. Greiner Honored.

Dr. A. B. Greiner, Rural Retreat, has been cited for teaching the adult Bible class of Grace Lutheran Church for fifty years. He was given a doctor's satchel containing fifty one-dollar bills. Dr. Greiner is eighty-six years of age and still practicing. He has been at Rural Retreat for fifty-seven years.

Dr. Mack I. Shanholtz,

Richmond, is the new president of the Association of State and Territorial Health Officers.

Symposium on Diabetes Mellitus.

A one-day symposium on Recent Advances in Diabetes Mellitus will be held at Georgetown University Medical Center, Washington, D. C., on March 8th. The following program will be presented: The Treatment of Diabetic Acidosis by Dr. James B. Field, Senior Investigator, National Institutes of Health, Bethesda; Vascular Disease in the Diabetic Patient by Dr. Charles A. Hufnagel, Professor of Experimental Surgery, Georgetown University; Ocular Manifestations of Diabetes by Dr. Henry Keen, Investigator, National Institutes of Health; Diabetic Neuropathy by Dr. Malcolm M.

Martin, Assistant Professor of Medicine, Georgetown University; Disordered Lipid Metabolism in Diabetes by Dr. Robert H. Williams, Professor of Medicine, University of Washington, Seattle; Hypolipemic Agents Used in Diabetic Patients by Dr. Edgar A. Haunz, Associate Professor of Medicine, University of North Dakota; Podiatric Care of the Diabetic by Dr. Edward L. Tarara, Podiatrist, Mayo Clinic, Rochester; Insulin Hypoglycemia by Dr. Sidney A. Tyroler, Instructor in Medicine, Georgetown University; The Treatment of Diabetes with Insulin by Dr. Haunz; The Use of the Sulfonylurea Drugs by Dr. L. Harvey Hamff, Clinical Associate Professor of Medicine, Emory University, Atlanta; Phenformin in Diabetic Management by Dr. Robert H. Williams; Dietary Treatment of Obese Diabetics by Dr. DeWitt E. DeLawter, Clinical Assistant Professor of Medicine, Georgetown University. A panel discussion and answer to all questions will be held in the afternoon.

This symposium is sponsored in part by a grant from Charles Pfizer and Company and all physicians are invited. There will be no registration fee.

Fairfax Hospital Staff.

Dr. J. D. Zylman, Arlington, has been elected president of the Fairfax County Hospital medical staff. Dr. Thomas B. McCord, Fairfax, is vice president; Dr. John C. Bauer, Arlington, secretary-treasurer; and Drs. Joseph Beinstein, Arlington, and Frances Ayres, Falls Church, members-at-large of the executive committee.

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Obituaries . . . .

Dr. John Mason Williams,

Prominent physician of Petersburg, died January 4th at the age of seventy-nine. He was a native of Prince George County and had practiced in Petersburg for more than fifty-five years. Dr. Williams graduated from the University College of Medicine in 1904 and practiced in Disputanta for several years. He served as surgeon for the Norfolk and Western Railroad.

Dr. Williams had been a member of The Medical Society of Virginia for fifty-six years and was made a Life Member in 1954.

His wife, three daughters and two sons survive him.

Dr. William Joshua Sturgis,

Beloved physician of Nassawadox, died December 20th following a short illness. He was eighty-two years of age and a graduate of the Medical College of Virginia in 1903. Dr. Sturgis had been a member of The Medical Society of Virginia since 1909.

The following resolutions were passed by the members of the Northampton-Accomac Memorial Hospital and the Northampton County Medical Society:

The people of The Eastern Shore of Virginia were greatly saddened on December 20, 1960, by the death of Dr. William J. Sturgis, Sr., of Nassawadox, Virginia. Dr. Sturgis was born in 1878. His college work was done at The College of William and Mary, and in 1903 he was graduated in medicine from The Medical College of Virginia. Returning to his home community, he began the practice of his profession.

He soon realized the extreme need for a community hospital where those who were sick and in distress could obtain the best care. While there were many others who gave of their time and talent, it was largely through his effort that on August 17, 1928, The Northampton-Accomack Memorial Hospital was formally opened. Throughout the remaining years of his life, his supreme interest was in the continued progress and improvement of this hospital in order that it might continue to be better equipped to serve the people of The Eastern Shore of Virginia.

He was a member of The Medical Society of Virginia and The American Medical Association; medical officer in The United States Public Health Service; member of The Northampton County Medical Society; and for a number of years served as Chairman of The Board of Trustees of The Northampton-Accomack Memorial Hospital. At a meeting of The Northampton Ruritans, on his 79th birthday, he was selected as the "Northampton Citizen of the Year". He was a member of the Red Bank Baptist Church, serving for many years as a member of The Board of Deacons.

He is survived by his widow, Mrs. Mary Sturgis; a daughter, Mrs. Douglas H. Crockett; and two sons, Dr. William J. Sturgis, Jr., and Mr. John Edward Sturgis. His older son, Dr. William J. Sturgis, Jr., is a member of the hospital surgical staff. He leaves also a sister, Miss Addie Sturgis.

The members of the hospital staff submit this tribute to his memory, realizing that the measure of a life is not its duration, but its donation. He will be greatly missed. Yet, his works will live after him to bless humanity.

Dr. W. T. Green, Jr.
Dr. J. Fred Edmunds
Committee

Whereas: God, in His infinite wisdom, has taken unto Himself His servant, our fellow physician, Dr. William J. Sturgis, Sr., and

Whereas: We recall with humility the unselfish motives which characterized his medical practice and devotion to the welfare of the entire Eastern Shore community, and

Whereas: We wish to honor his memory, therefore

Be It Resolved: That we bow in humble submission to the will of the Almighty, that we strive to emulate his example, and that we cause this expression of our regard to be spread upon the permanent records of the Northampton County Medical Society, of The Medical Society of Virginia, and that a copy be sent to his bereaved family.

John R. Hamilton
H. L. Denoon, Jr.
W. Carey Henderson, *Chairman*

Dr. Greenville Ramsey Berkeley,

Well known physician of Norfolk, died December 7th. He was seventy-seven years of age and received his medical degree from the Jefferson Medical College in 1960. He located in Norfolk the following year. Dr. Berkeley was formerly medical examiner and city jail physician. He retired from active practice in 1954, as a result of a fractured pelvis received the year before which proved a severe handicap to him.

Dr. Berkeley had been a member of The Medical Society of Virginia since 1912.

He is survived by a son and a daughter.

Dr. Thomas Nathaniel Spessard,

Norfolk neuro-psychiatrist, died December 14th. He was fifty-seven years of age and graduated from the Medical College of Virginia in 1927. Dr. Spessard was a captain in the U. S. Navy Reserve Medical Corps. He had been a member of The Medical Society of Virginia for thirty-one years.

His wife and two daughters survive him.

Dr. Thomas Edward Jones,

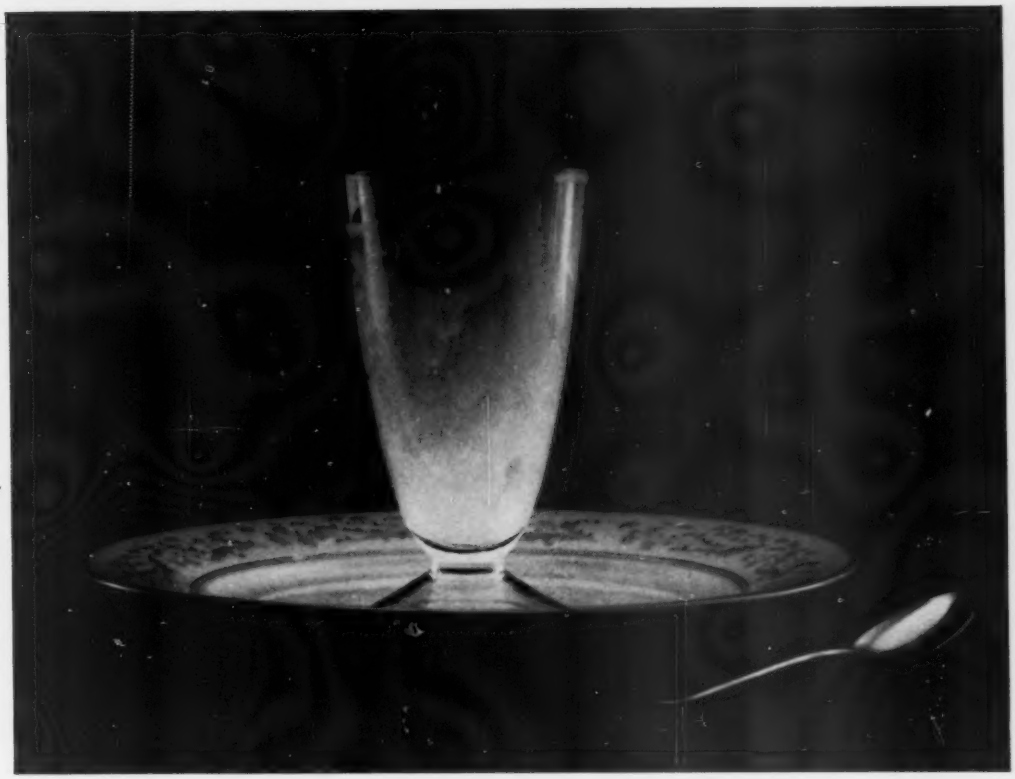
Charlottesville, died December 19th, at the age of seventy-five. He was a graduate in medicine from the University of Virginia in 1909. Before he came to Charlottesville, Dr. Jones practiced in Sioux Falls, South Dakota, and Lynchburg. He had been in Charlottesville for twenty years. Dr. Jones was a member of The Medical Society of Virginia, having joined in 1938.

He is survived by two daughters and one son.

Dr. James Terrell May, Jr.

Norfolk, died December 11th, death being due to asphyxiation during a fire in his apartment. He was forty-one years of age and a graduate of the Medical College of Virginia in 1945. Dr. May was a member of The Medical Society of Virginia.

He is survived by his wife, two daughters and four sons.



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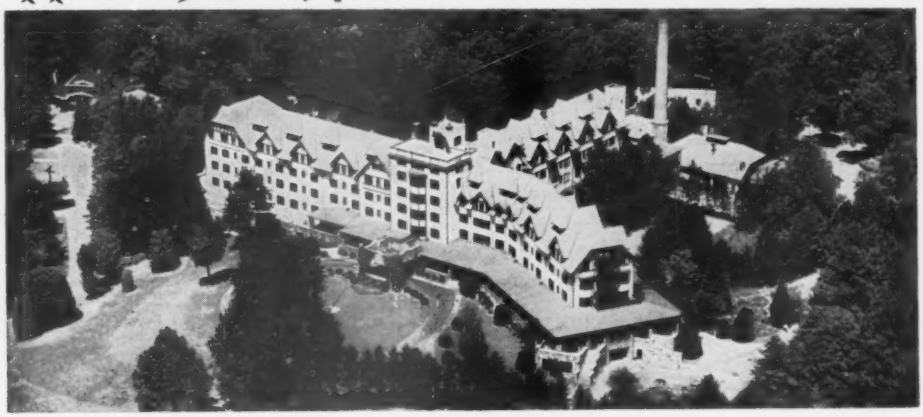

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Pyridoxine Hydrochloride	2 mg.
Cobalamin (Vitamin B_{12})	2 mcg.
Calcium Pantothenate	5 mg.
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Calcium Pantothenate	20 mg.
Ascorbic Acid	200 mg.

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Calcium Pantothenate	10 mg.
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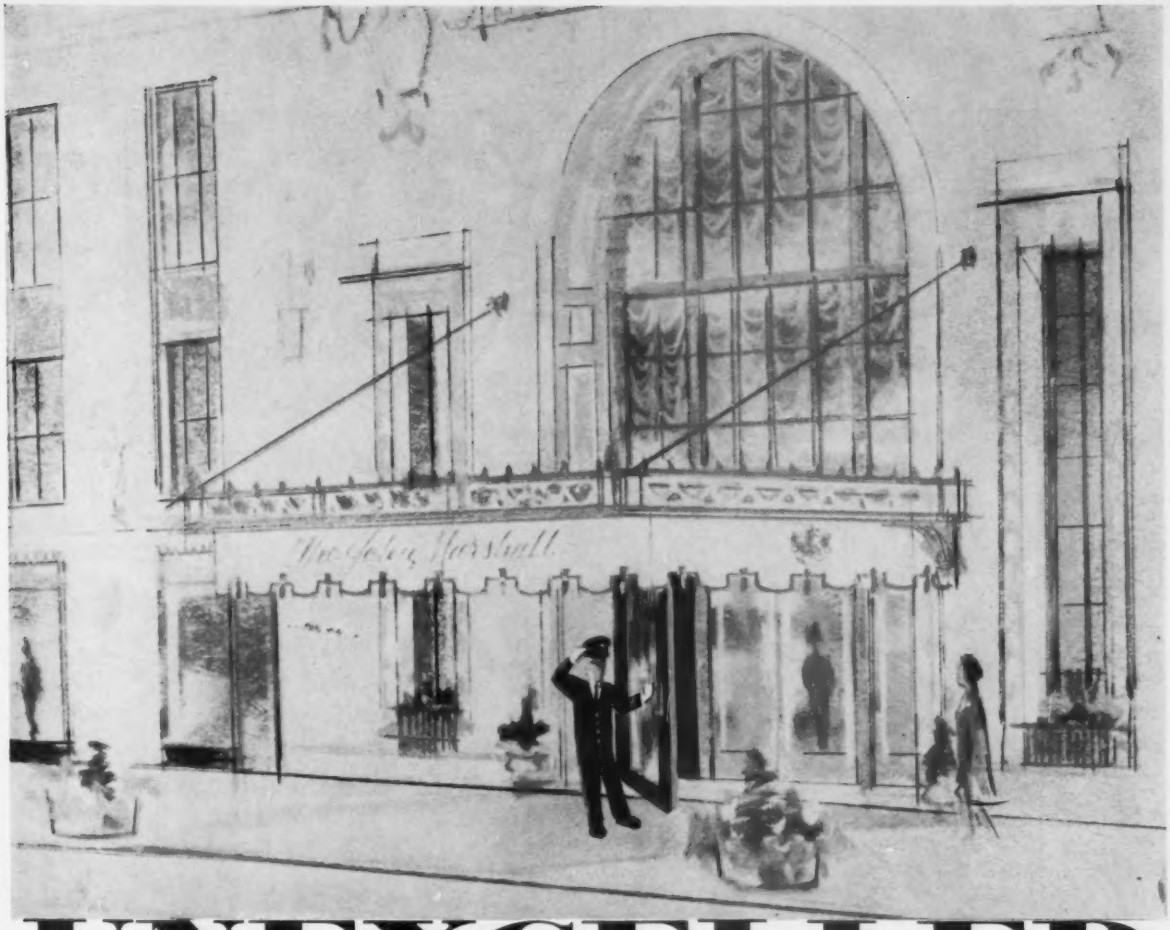

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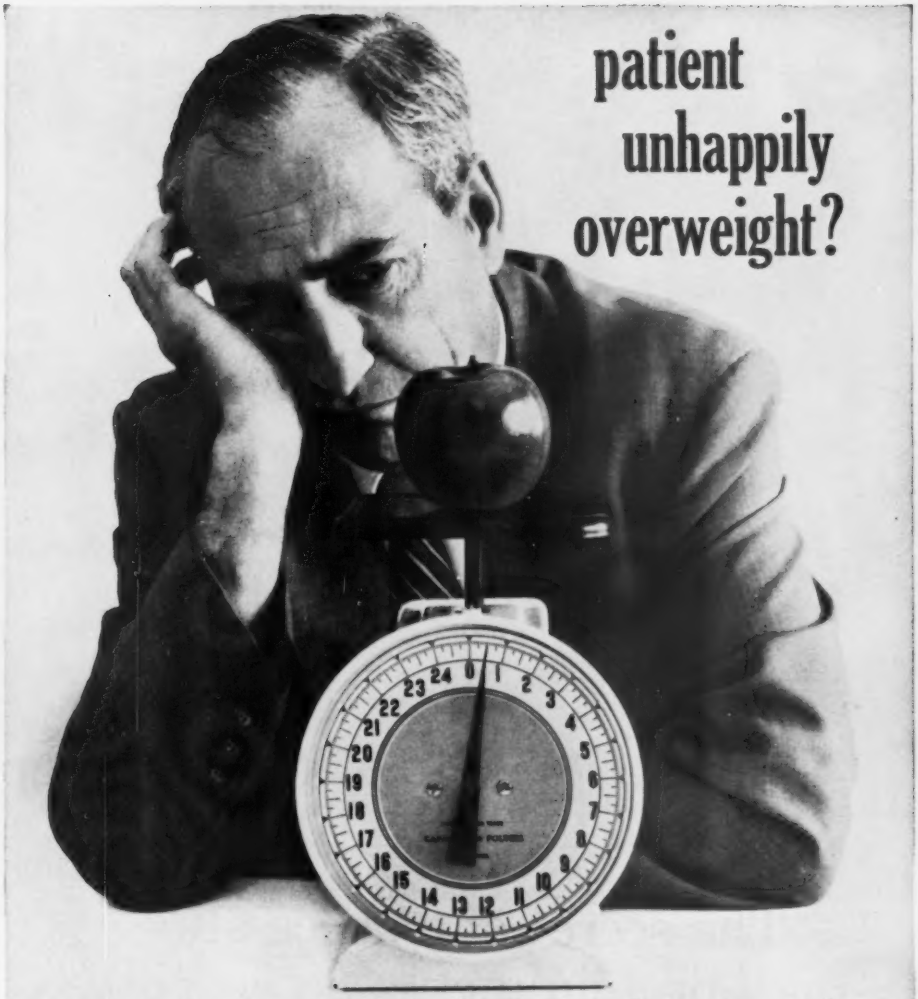

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Supplied: Tablets 5 mg., scored. Bottles of 100 and 1000.

[1] Douglas, H. S.: West. J. Surg. 59:238 (May) 1951.

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Flavorful fare your patient will welcome!

The secret of a successful high protein diet is acceptance

The acceptance of *any* diet depends on its appetite appeal. Your high protein diet patients should find these dishes both tempting—*and* economical . . . like the fluffy omelet above, folded over penny-sliced frankfurters. Ground meat, flaked fish or cheese are also rich (but inexpensive) sources of protein.

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Protein, 0.8 grm.; calories, 104/8 oz. glass (Average of American Beers)

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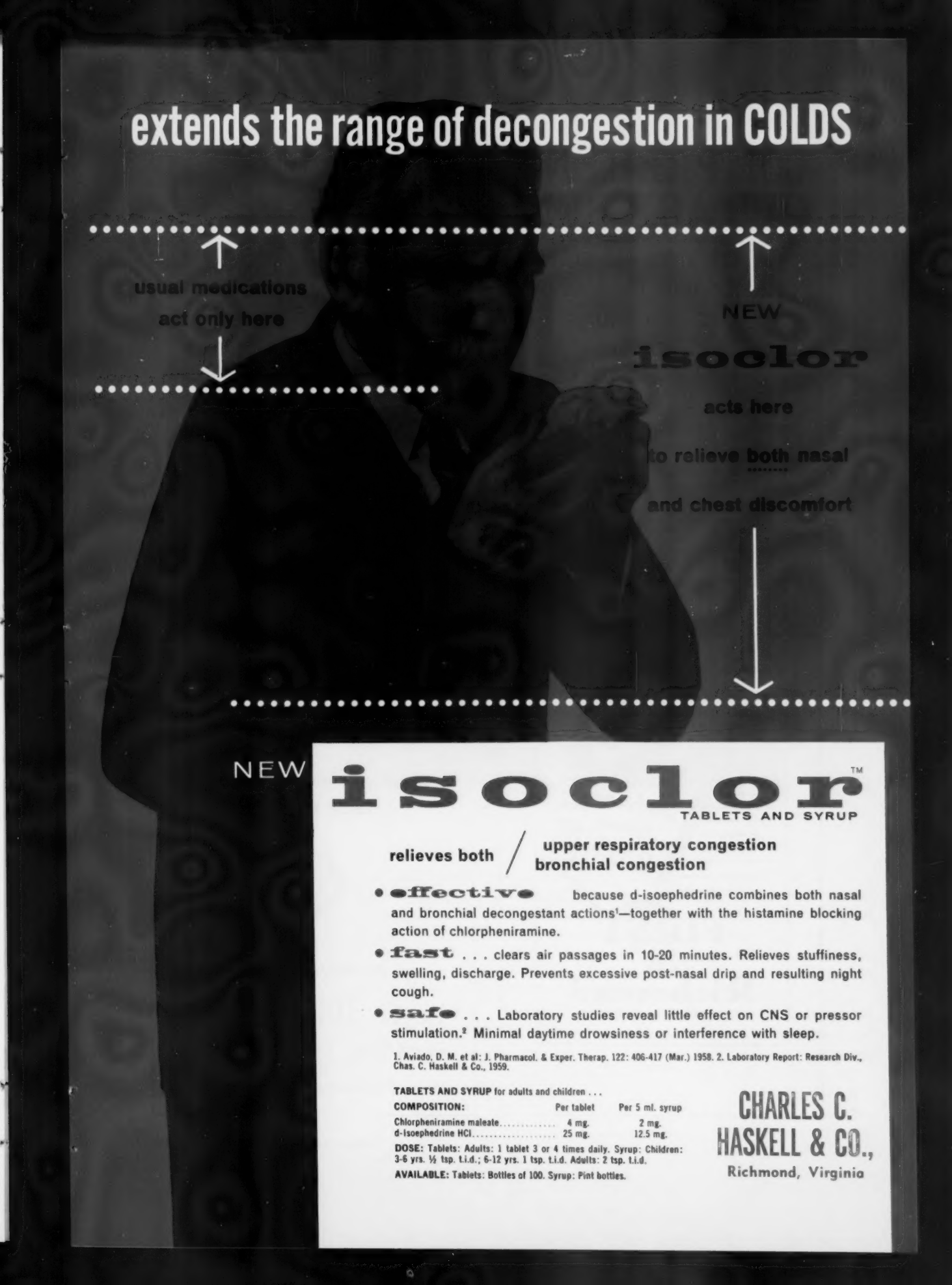
extends the range of decongestion in COLDS



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TABLETS AND SYRUP

relieves both / **upper respiratory congestion bronchial congestion**

- **effective** because d-isoephedrine combines both nasal and bronchial decongestant actions[1]—together with the histamine blocking action of chlorpheniramine.
- **fast** . . . clears air passages in 10-20 minutes. Relieves stuffiness, swelling, discharge. Prevents excessive post-nasal drip and resulting night cough.
- **safe** . . . Laboratory studies reveal little effect on CNS or pressor stimulation.[2] Minimal daytime drowsiness or interference with sleep.

1. Aviado, D. M. et al: J. Pharmacol. & Exper. Therap. 122: 406-417 (Mar.) 1958. 2. Laboratory Report: Research Div., Chas. C. Haskell & Co., 1959.

TABLETS AND SYRUP for adults and children . . .

COMPOSITION:	Per tablet	Per 5 ml. syrup
Chlorpheniramine maleate	4 mg.	2 mg.
d-Isoephedrine HCl	25 mg.	12.5 mg.

DOSE: Tablets: Adults: 1 tablet 3 or 4 times daily. Syrup: Children: 3-6 yrs. ½ tsp. t.i.d.; 6-12 yrs. 1 tsp. t.i.d. Adults: 2 tsp. t.i.d.

AVAILABLE: Tablets: Bottles of 100. Syrup: Pint bottles.

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- ▲ brightens the outlook
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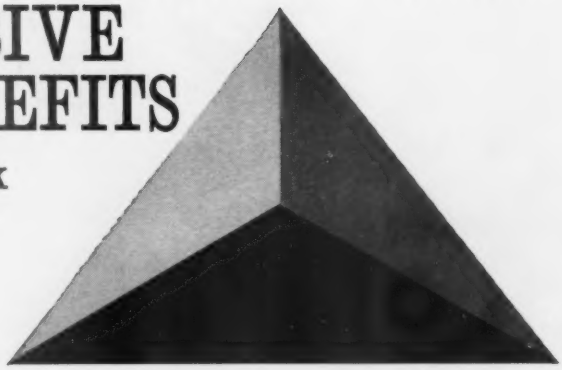

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Each capsule contains: Ethinyl Estradiol 0.01 mg. • Methyl Testosterone 2.5 mg. • d-Amphetamine Sulfate 2.5 mg. • Vitamin A (Acetate) 5,000 U.S.P. Units • Vitamin D 500 U.S.P. Units • Vitamin B_{12} with AUTRINIC® Intrinsic Factor Concentrate 1/15 U.S.P. Unit (Oral) • Thiamine Mononitrate (B_1) 5 mg. • Riboflavin (B_2) 5 mg. • Niacinamide 15 mg. • Pyridoxine HCl (B_6) 0.5 mg. • Calcium Pantothenate 5 mg. • Folic Acid 0.4 mg. • Choline Bitartrate 25 mg. • Inositol 25 mg. • Ascorbic Acid (C) as Calcium Ascorbate 50 mg. • l-Lysine Monohydrochloride 25 mg. • Vitamin E (Tocopherol Acid Succinate) 10 Int. Units • Rutin 12.5 mg. • Ferrous Fumarate (Elemental Iron, 10 mg.) 30.4 mg. • Iodine (as KI) 0.1 mg. • Calcium (as $CaHPO_4$) 35 mg. • Phosphorus (as $CaHPO_4$) 27 mg. • Fluorine (as CaF_2) 0.1 mg. • Copper (as CuO) 1 mg. • Potassium (as K_2SO_4) 5 mg. • Manganese (as MnO_2) 1 mg. • Zinc (as ZnO) 0.5 mg. • Magnesium (MgO) 1 mg. • Boron (as $Na_2B_4O_7.10H_2O$) 0.1 mg. Bottles of 100, 1000.

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Each of the babies pictured on this page was borne by a mother with a *documented* previous history of true habitual abortion, who was treated with DELALUTIN during the pregnancy leading to this birth

LIVING PROOF OF FETAL SALVAGE WITH

DELALUTIN

SQUIBB HYDROXYPROGESTERONE CAPROATE

Improved Progestational Therapy

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DELALUTIN offers these advantages over other progestational agents

• long-acting sustained therapy • more effective in producing and maintaining a completely matured secretory endometrium • no androgenic effect • more concentrated solution requiring injection of less vehicle • unusually well-tolerated, even in large doses • fewer injections required • low viscosity makes administration easy

Complete information on administration and dosage is supplied in the package insert

Supply:
Vials of 2 and 10 cc., each containing 125 mg. of hydroxyprogesterone caproate in benzyl benzoate and sesame oil.

Also available: DELALUTIN 2X in 5 cc. multiple-dose vials. Each cc. contains 250 mg. hydroxyprogesterone caproate in castor oil, preserved with benzyl alcohol.

SQUIBB *Squibb Quality—The Priceless Ingredient*

"DELALUTIN"® IS A SQUIBB TRADEMARK

in sinusitis, colds
and upper respiratory
disorders

NEW

DIMETAPP® Extentabs®

let your patients
breathe easier!

In sinusitis, colds and other upper respiratory and allergic disorders, new DIMETAPP Extentabs offer more useful decongestant therapy. Stuffiness, drip and other annoying symptoms of congestion are effectively relieved with minimum side effects.

UNSURPASSED RELIEF OF NASAL CONGESTION DIMETAPP Extentabs contain an unexcelled antihistamine, Dimetane, which has produced good to excellent results in thousands of cases of allergic respiratory disorders.* In DIMETAPP Extentabs, the action of Dimetane with two outstanding decongestants—phenylephrine and phenylpropanolamine—promptly dries secretions and reduces edema and congestion in the nose, the sinuses, and the upper respiratory tract.

CLEAR BREATHING FOR 12 HOURS ON 1 TABLET Long-acting DIMETAPP Extentabs offer up to 12-hour relief on just one tablet. Easier to use than nose drops or sprays, DIMETAPP reaches into areas topical decongestants can't touch—without rebound congestion.

EXCEPTIONAL FREEDOM FROM SIDE EFFECTS With DIMETAPP Extentabs, there's little problem of either drowsiness or overstimulation. The antihistamine component, Dimetane, offers a high percentage of effective relief with only drowsiness as a possible infrequent side effect.* Small, fully efficient dosages of decongestants minimize the danger of overstimulation.

DIMETAPP Extentabs contain Dimetane® (parabromdylamine [brompheniramine] maleate) 12 mg., phenylephrine HCl 15 mg., and phenylpropanolamine HCl 15 mg. Dependable Extentabs construction assures relief of symptoms for up to 12 hours with 1 tablet.

Dosage: Adults—1 Extentab q. 8-12 hours. Children over 6—1 Extentab q. 12 hours. Administer with caution to patients with cardiac or peripheral vascular diseases and hypertension, and to those sensitive to antihistamines. See package insert for further details. **Supplied:** bottles of 100 and 500.

*Full bibliography on Dimetane available on request.

A. H. ROBINS CO., INC. Richmond 20, Virginia
Ethical Pharmaceuticals of Merit Since 1878

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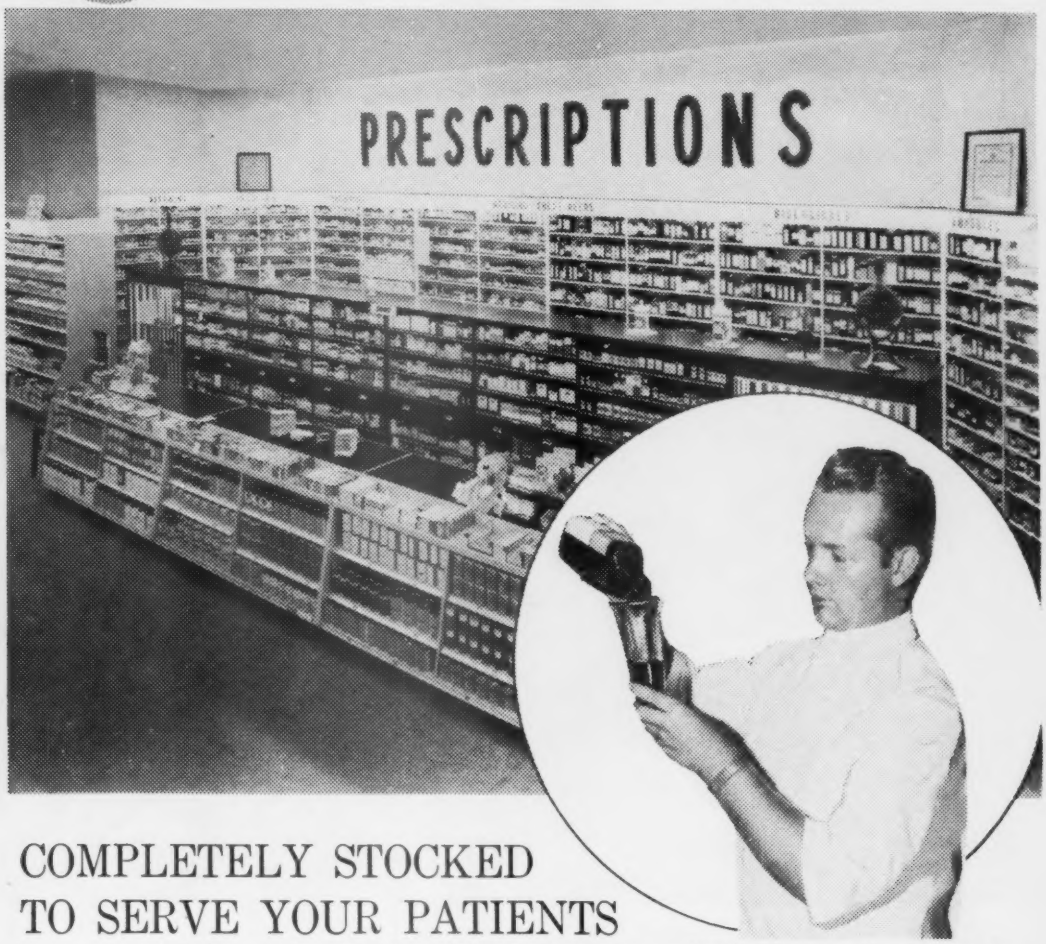

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TO SERVE YOUR PATIENTS

We work closely with the pharmaceutical manufacturers to insure having the newest drugs in stock as soon as you prescribe them for your patients. New drugs are received in our prescription departments at the rate of more than one a day.

Our prescription inventories are carefully checked every month to eliminate old or outdated drugs from our stock for the protection of your patients.

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Stores to serve you in Virginia!

MICROBIOLOGICAL AND PHARMACOLOGICAL PROPERTIES

In vitro studies show that STAPHCILLIN is a bactericidal penicillin with activity against staphylococci resistant to penicillin G. Strains of staphylococci so far tested have been sensitive to STAPHCILLIN *in vitro* at concentrations of 1-6 mcg. per ml. These levels are readily attained in the blood and tissues by administration of STAPHCILLIN at the recommended dosage. This unique attribute is probably due to the fact that STAPHCILLIN is stable in the presence of staphylococcal penicillinase. STAPHCILLIN also resists degradation by *B. cereus* penicillinase. The antimicrobial spectrum of STAPHCILLIN with regard to other microorganisms is qualitatively similar to that of penicillin G; but considerably higher concentrations of STAPHCILLIN are required for bactericidal activity than is the case with penicillin G.

STAPHCILLIN is rapidly absorbed after intramuscular injection. Peak blood levels (6-10 mcg./ml. on the average after a 1.0 Gm. dose) are attained within 1 hour; and then progressively decline to less than 1 mcg. over a 4 to 6 hour period. It is poorly absorbed from the gastrointestinal tract. STAPHCILLIN is rapidly excreted by the kidney.

As shown by animal studies, STAPHCILLIN is readily distributed in body tissues after intramuscular injection. Of the tissues studied, highest concentrations are reached in the kidney, liver, heart and lung in that order; the spleen and muscles show lower concentrations of the antibiotic. STAPHCILLIN diffuses into human pleural and prostatic fluids, but its diffusion into the spinal fluid has not yet been completely studied. However, one patient with meningitis showed a significant concentration in his spinal fluid while on STAPHCILLIN therapy.

Toxicity studies with STAPHCILLIN and penicillin G in animals show that they have approximately the same low order of toxicity.

Certain staphylococci can be made resistant to STAPHCILLIN in the laboratory, but this resistance is not related to their penicillinase production. During the clinical trials, no STAPHCILLIN-resistant strains of staphylococci were observed or developed; the possibility of the emergence of such strains in the clinical setting awaits further observation.

PRECAUTIONS

During the clinical trials, several mild skin reactions, e.g., itching, papular eruption and erythema were observed both during and after discontinuance of STAPHCILLIN therapy. Patients with histories of hay fever, asthma, urticaria and previous sensitivity to penicillin are more likely to react adversely to the penicillins. It is important that the possibility of penicillin anaphylaxis be kept in mind. Epinephrine and the usual adjuvants (antihistamines, corticosteroids) should be available for emergency treatment. Because of the resistance of STAPHCILLIN to destruction by penicillinase, parenteral *B. cereus* penicillinase may not be effective for the treatment of allergic reactions. Information with regard to cross-allergenicity between penicillin G, penicillin V, phenethicillin (Syncillin) and STAPHCILLIN is not available at present. If superinfection due to Gram-negative organisms or fungi occurs during STAPHCILLIN therapy, appropriate measures should be taken.

SUPPLY

List 79502 — 1.0 Gm. dry filled vial.

BRISTOL LABORATORIES • SYRACUSE, NEW YORK
Division of Bristol-Myers Company

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UNIQUE SYNTHETIC "STAPH-CIDAL" PENICILLIN

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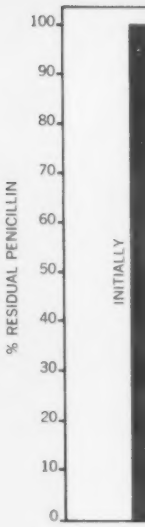

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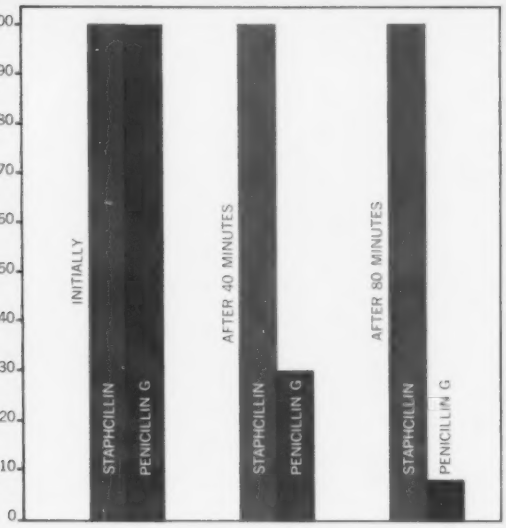

In the presence of staphylococcal penicillinase, STAPHCILLIN remained active and retained its antibacterial action. By contrast, penicillin G was rapidly destroyed in the same period of time. *(After Gourevitch et al., to be published)*

pecifically for "resistant" staph...

Staphcillin™

sodium dimethoxyphenyl penicillin
FOR INJECTION

e failure of staphylococcal infections to respond to penicillin therapy is attributed to penicillin-destroying enzyme, penicillinase, produced by the invading staphylococcus.

like other penicillins:

STAPHCILLIN is effective because it retains its antibacterial activity despite the pres-ce of staphylococcal penicillinase.

The clinical effectiveness of STAPHCILLIN has been confirmed by dramatic results in vide variety of infections due to "resistant" staphylococci, many of which were serious d life-threatening.

ke other penicillins:

STAPHCILLIN has no significant systemic toxicity. It is well tolerated locally, and in or irritation at the injection site is comparable to that following the injection of nicillin G. *In occasional cases, typical penicillin reactions may be experienced.*

PROFESSIONAL INFORMATION SERVICE – The attached Official Package Circular provides complete information on the indications, dosage, and precautions for the use of STAPHCILLIN. If you desire additional information concerning clinical experiences with STAPHCILLIN, the Medical Department of Bristol Laboratories is at your service. You may direct your inquiries via collect telephone call to New York, PLaza 7-7061, or by mail to Medical Department, Bristol Laboratories, 630 Fifth Ave., N. Y. 20, N. Y.

RISTOL LABORATORIES • SYRACUSE, NEW YORK

Division of Bristol-Myers Company

ANNOUNCING—
SPECIFICALLY FOR
INFECTIONS DUE TO
"RESISTANT" STAPHYLOCOCCI

AN ENTIRELY NEW SYNTHETIC
"STAPH-CIDAL" PENICILLIN

Staphcillin

sodium dimethoxyphenyl penicillin
FOR INJECTION

UNIQUE—BECAUSE IT
RETAINS ANTIBACTERIAL
ACTIVITY IN THE PRESENCE OF
STAPHYLOCOCCAL PENICILLINASES
WHICH INACTIVATE
OTHER PENICILLINS

Bristol™

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NEW SYNTHETIC PENICILLIN FOR "RESISTANT" STAPH

OFFICIAL PACKAGE CIRCULAR
November, 1960

STAPHCILLIN™

(sodium dimethoxyphenyl penicillin)

For Injection

DESCRIPTION

STAPHCILLIN is a unique new synthetic parenteral penicillin produced by Bristol Laboratories for the specific treatment of staphylococcal infections due to resistant organisms. Its uniqueness resides in its property of resisting inactivation by staphylococcal penicillinase. It is active against strains of staphylococci which are resistant to other penicillins.

Each dry filled vial contains: 1 Gm. STAPHCILLIN (sodium dimethoxyphenyl penicillin), equivalent to 900 mg. dimethoxyphenyl penicillin activity.

INDICATIONS

STAPHCILLIN is recommended as specific therapy only in infections due to strains of staphylococci resistant to other penicillins, e.g.:

Skin and soft tissue infections: cellulitis, wound infections, carbuncles, pyoderma, furunculosis, lymphangitis and lymphadenitis.

Respiratory infections: staphylococcal lobar or bronchopneumonia, and lung abscesses combined with indicated surgical treatment.

Other infections: staphylococcal septicemia, bacteremia, acute or subacute endocarditis, acute osteomyelitis and enterocolitis.

Infections due to penicillin-sensitive staphylococci, streptococci, pneumococci and gonococci should be treated with Syncillin® or parenteral penicillin G rather than STAPHCILLIN. Treponemal infections should be treated with parenteral penicillin G.

DOSAGE AND ADMINISTRATION

STAPHCILLIN is well tolerated when given by deep intragluteal or intravenous injection.

As is the case with other antibiotics, the duration of therapy should be determined by the clinical and bacteriological response of the patient. Therapy should be continued for at least 48 hours after the patient has become afebrile, asymptomatic and cultures are negative. The usual duration has been 5-7 days.

Intramuscular route: The usual adult dose is 1 Gm. every 4 or 6 hours. Infants' and children's dosage is 25 mg. per Kg. (approximately 12 mg. per pound) every 6 hours.

Intravenous route: 1 Gm. every 6 hours using 50 ml. of sterile saline solution at the rate of 10 ml. per minute.

**Warning:* Solutions of STAPHCILLIN and kanamycin should not be mixed, as they rapidly inactivate each other. Data on the results of mixing STAPHCILLIN with other antibiotics are being accumulated.

DIRECTIONS FOR RECONSTITUTION

Add 1.5 ml. sterile distilled water or normal saline to a 1 Gm. vial and shake vigorously. Withdraw the clear, reconstituted solution (2.0 ml.) into a syringe and inject. The reconstituted solution contains 500 mg. of STAPHCILLIN per ml. Reconstituted solutions are stable for 24 hours under refrigeration.

For intravenous use, dilute the reconstituted dose in 50 ml. of sterile saline and inject at the rate of 10 ml. per minute.

*This statement supersedes that in the Official Package Circulars dated September and/or October, 1960.

(continued)

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EFFECTIVE ANTIHISTAMINE-DECONGESTANT ACTION

COVANAMINE'S combination of two antihistamines and two decongestants—shrinks swollen turbinates—opens blocked ostia—promotes drainage—reestablishes patency—in the treatment of common colds, rhinitis, sinusitis, nasal allergies and post nasal drip.

Constant therapeutic levels are maintained by COVANAMINE'S Sustained Action Tablets which meter out the active ingredients . . . with minimal side effects, less drowsiness.

COVANAMINE (TM)

Sustained Action Tablets

Also available as Black Cherry Flavored COVANAMINE LIQUID; COVANAMINE EXPECTORANT provides the liquid formula plus glyceryl guaiacolate.

Each Sustained Action (continuous release) COVANAMINE tablet contains: phenylephrine HCl 15 mg., phenylpropanolamine HCl 25 mg., Chlorpheniramine maleate 4 mg., and pyrilamine maleate 25 mg.

COVANAMINE LIQUID provides ¼ the tablet formula in each 5 ml. teaspoon.

COVANAMINE EXPECTORANT provides the liquid formula plus glyceryl guaiacolate 100 mg. per teaspoon.

Dosage: Tablets: Adults—1 tablet (swallowed without chewing) morning, mid-afternoon and at bedtime; Children, 6 to 12 years—½ tablet. Liquid and Expectorant: Adults—2 teaspoonfuls every four hours. Children 6 to 12 years—1 teaspoonful every four hours; 1 to 6 years—½ teaspoonful every four hours; under 1 year—¼ teaspoonful every four hours.

VANPELT AND BROWN, INC. Richmond, Virginia

Put your low-back patient back on the payroll

Soma's prompt relief of pain and stiffness can get your low-back patients back to work in days instead of weeks

Soma is unique because it combines the properties of an effective muscle relaxant and an independent analgesic in *a single drug.* Unlike most other muscle relaxants, which can only relax muscle tension, Soma attacks both phases of the pain-spasm cycle at the same time.

Thus with Soma, you can break up both pain and spasm fast, effectively . . . help give your patient the two things he wants most: relief from pain and rapid return to full activity.

Soma is notably safe. Side effects are rare. Drowsiness may occur, but usually only with higher dosages. Soma is available in 350 mg. tablets. Usual dosage is 1 tablet q.i.d.

The muscle relaxant with an independent pain-relieving action

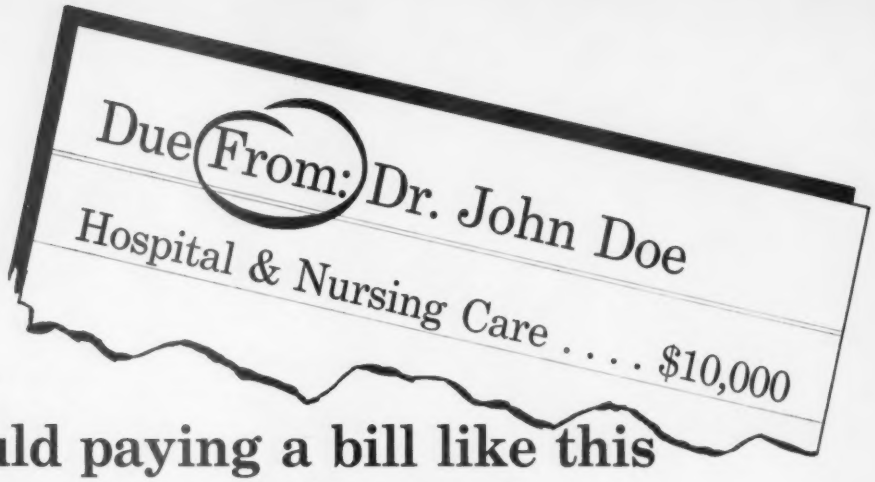
SOMA®

(carisoprodol, Wallace)

Wallace Laboratories, Cranbury, New Jersey

How you can help save your patients a month's pay

Kestler reports in J.A.M.A. (April 30, 1960) that conventionally treated low-back syndrome patients required an average of 41 days for full recovery (range: 3 to 90 days). The addition of Soma therapy in this comparative investigation reduced the average to 11.5 days (range: 2 to 21 days). With Soma, patients averaged full recovery 30 days sooner.



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- **What would paying a bill like this do to <u>your</u> personal finances?**
- **And what about additional bills for your continuing Office Expenses — if YOU had been the <u>patient</u>?**

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. . . knowing that today's hospital confinements mean BIG bills, you should be the first to own "catastrophic" hospital-nurse insurance for yourself and your family's *assured* protection.

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. . . knowing that today it costs BIG money to operate your office — even when you are sick or injured and *can't* be 'on duty' — it's only good business to obtain Overhead Expense protection.

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Professional Overhead Expense

PAYS covered Office Expenses — Rent, Employees' Salaries, Heat, etc. — when you are continuously disabled by injury or sickness for 14 days or more. Payments are made *directly to you*, and can continue for as long as 1 year if you are totally disabled that length of time.

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UNDERWRITTEN BY AMERICAN CASUALTY CO. READING, PA.

DAVID A. DYER, Administrator

Medical Arts Building **Roanoke, Virginia**

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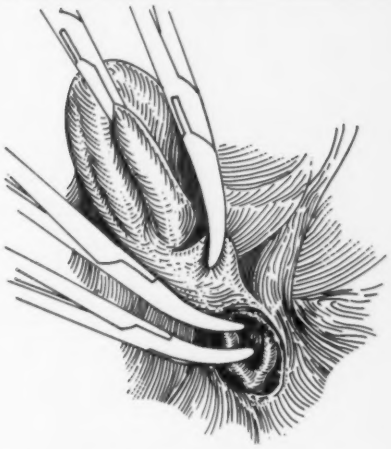

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Source: Farris, J. M., and Smith, G. K.: M. Clin. North America *43*:1133 (July) 1959.

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